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Monthly Message

Last year, some members of the Medical Advisory Council of the Canadian Department of National Defence joined with the Civilian Advisory Council of this office and visited Maxwell and Gunter Air Force Bases and the Naval Air Training Station at Pensacola. The group consisted of Dr. Joseph A. MacFarlane, Chairman of the Council and Dean of the University of Toronto Faculty of Medicine; Dr. Ian Macdonald, Professor of Medicine at Toronto; Brigadier Kenneth A. Hunter, RCAMC, the Executive Director of the Council; and Air Commodore Alexander A. G. Corbet, RCAF.

This trip was so successful that in March 1958 the two groups joined again to visit Sandia Base and Holloman Air Force Base, New Mexico, and Edwards Air Force Base, California. This time, in addition to our own Council, the group consisted of Dr. MacFarlane, Dr. Alfred W. Farmer, Dr. Desmond Magna, and Air Commodore Corbet; and a most cordial reception was accorded everywhere.

During these visits our guests sit with us at one of the regular meetings of our Advisory Council and our programs and problems are discussed together.

In the summer of 1957 on a trip to the Far Northeast a group of us, while at Goose Bay, took the liberty of visiting the Canadian Armed Forces Hospital on the other side of the airfield. At that time both the Director and Deputy Director were away, the latter a surgical patient in the U. S. Air Force Hospital at Goose Bay. Nevertheless we received a most cordial reception from the lone medical officer still left on duty, a 1957 graduate of Dalhousie University Faculty of Medicine at Halifax.*

In September 1956 I was the guest of the Canadian Advisory Council at one of its meetings in Ottawa. A continuation of these mutually beneficial exchange visits is planned by both groups. During World War II in both the Mediterranean and the European Theatres we were closely associated, first in England and then in Italy, France, and Germany; and in 1950 a conference on medical supplies and standardization of equipment was held in Montreal including representatives from the United Kingdom,

*Requires one year of internship before M. D. degree.

Canada, and the United States, which I was privileged to attend. Unfortunately, little came of that meeting, but we hope that the exchange visits so recently inaugurated will continue with increasing success.

Frank B. Berry

FRANK B. BERRY, M. D.
Assistant Secretary of Defense
(Health and Medical)

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Foreword

The *United States Armed Forces Medical Journal* is the medium for disseminating information of administrative and professional interest to all medical personnel of the Department of Defense. The Assistant Secretary of Defense (Health and Medical) and the Surgeons General of the several services invite all medical officers, dental officers, Medical Service Corps officers, Nurse Corps officers, and officers of the Veterinary Corps of the Armed Forces, and the medical consultants of the Army, Navy, and Air Force to submit manuscripts for publication in this *Journal*.

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SEVERE DYSBARISM IN AIR FORCE OPERATIONS AND TRAINING

CHARLES A. BERRY, *Major, USAF (MC)*

THE term dysbarism is used to define the symptom complex resulting from exposing the body to reduced atmospheric pressure. It includes both trapped and evolved gas syndromes, and is distinctly separate from hypoxia. Decompression sickness is usually synonymous with severe dysbarism and neurocirculatory collapse.

The advent of jet aircraft with their altitude-hungry engines has created a large population exposed to a significant decrease in atmospheric pressure in both the training and the operational situation. The number of persons exposed or potentially exposed to altitudes in excess of 30,000 feet will be further increased by the advent of all jet aircraft units in the U. S. Air Force and civilian jet airliners. Inevitably this increased exposure will lead to more cases of severe dysbarism.

OPERATIONAL PROBLEM

The problem of severe dysbarism in operational aircraft has been highlighted in recent years by several deaths. Two cases in the U. S. Air Force have been reported by Haymaker, Johnston, and Downey[1] and mention was made of another. A fourth report has been received.[2] Fryer[3] described three deaths in the Royal Air Force, occurring after aircraft flight and as a result of decompression sickness. In addition to these deaths there are numerous cases of severe dysbarism in persons who fortunately recovered.[4-6, 15, 16] The aviation medicine consultation service at

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Presented at the Second European Congress of Aviation Medicine, Stockholm, Sweden, September 1957.

From School of Aviation Medicine, U. S. Air Force, Randolph Air Force Base, Tex.

this school recently has studied two patients and received reports of six others, all of whom suffered severe dysbarism in operational aircraft. One of these persons[5] exhibits findings of a "permanent" residual from his dysbarism episode. The pertinent data concerning these cases are summarized in table 1. There has been conflicting opinion concerning the possibility of permanent residual from decompression sickness. Some authors have stated the only known evidence was a dog with residual paralysis of the hind legs,[4] but this was prior to the report by Döbeln and Höök[7] which described a chamber reactor with residual paralysis. In the U. S. Air Force case the patient shows damage of the left choroid and severe damage to the visual pathways manifested by bilaterally impaired color vision fields. This residual is called permanent on the basis that it existed from January 1953 to October 1954, when the patient was last seen. His present status is unknown.

TABLE 1. *Severe dysbarism occurring in flight in the Air Force, 1953-1955*

Case No.	Date	Type of aircraft	Cabin altitude (feet)	Time at altitude	Clinical findings
1	Feb. 1953	T-33	38,000 (rapid decompression)	0 min.	Loss of feeling, right side; loss of muscular control, right side; blurred vision; damage of left choroid; white-field defect, left eye; color-field defect, both eyes.
2	18 Sept. 1953	B-45	30,000	30 min.	Bends, confusion, paresthesia, nausea and vomiting, sweating, ataxia, astereognosis, and positive Romberg.
3	29 Apr. 1956	T-33	31,000	1 hr.	Bends, fatigue, fainted; in state of shock, 12 hr; pericarditis.
4	25 Nov. 1956	T-33	24,000	1 hr.	Visual blurring, bends, chokes, paresthesia, headache, weak right hand, unsteady gait.
5	8 Feb. 1957	T-33	30,000	15 min.	Bends in elbow, wrist, and knees; headache; blurred vision.
6	5 Dec. 1955	B-47	37,000 (rapid decompression)	10 min.	Bends, dizziness, blurred vision.
7	14 July 1952	T-33	30,000-32,000	20 min.	Bends, paresthesia, visual blurring, delayed neurologic findings and confusion.
8	30 Nov. 1955	B-29	35,000	30 min.	Headache, diplopia, dizziness, slurred speech.

The eight cases of severe dysbarism in flight listed are not of great import when considered in the light of the hundreds of thousands of flying hours completed without incident in the same period. I believe, however, that many unreported cases exist, and that the cost of an aircraft and the investment in a trained pilot make even one such incident a very undesirable and costly affair.

It is of interest to note that five of the eight reported incidents occurred in T-33 aircraft. Rapid or explosive decompression is of great concern in our high-altitude, high-performance aircraft

for it exposes the crew to reduced atmospheric pressure and at the same time increases the risk of hypoxia and dysbarism by its sudden onset. In a recent compilation of data on some 98 rapid decompressions, in the U. S. Air Force, occurring over a 52-month period there were no instances of decompression sickness noted.[8] Only 32 of these decompressions occurred at altitudes in excess of 30,000 feet, and of these only 8 were in bomber-type aircraft. The fighters all descended rapidly and immediately, thus further reducing the risk of decompression sickness. Two cases listed in table 1 (cases 1 and 6) apparently were not reported to the Air Force Directorate of Flight Safety, but these men also had rapid decompression during this reporting period and they developed decompression sickness. In this regard it should be noted that frequently the T-33 is flown with the pressurization system functioning only well enough to lower the cabin altitude 1,000 or 2,000 feet below ambient. This is much more dangerous than rapid decompression for there is no emergency development of difficulty; instead, the insidious exposure to low atmospheric pressure exists during the entire flight plan.

Incidents of severe in-flight dysbarism are not uniformly reported in the Air Force. The only sources of such information are cases reported in the literature, by physiological training officers, flight surgeons, or the aviation medicine consultation service. In order to gather more data on this subject, this school has initiated proceedings to require reporting of these in-flight or operational cases as we do the chamber reactors.

TRAINING PROBLEM

In order to fly, or even ride as a passenger, in our high-altitude, high-performance aircraft, Air Force personnel must participate in a physiologic training course.[9] The curriculum of this course includes an altitude chamber flight to 43,000 feet. A generally standardized flight pattern is used by the various training units. As a result of the large number of personnel exposed to altitude in this program, a certain number of severe dysbarism cases (chamber reactors) are seen.

In the U. S. Air Force physiologic training program the various grades of chamber reactions are defined and reported as follows:

Grade I—Any slight unnatural sensation.
Grade II—Slight to moderate pain not requiring removal from or interruption of the chamber flight.
Grade III—Moderate to severe pain or other reactions requiring removal from the chamber.
Grade IV—Any chamber reaction requiring admission to a hospital.

In the Air Force during the years 1950 through 1955 there were 125 Grade IV chamber or dysbarism reactions reported.

The occurrence of the various symptom grades which precede the Grade IV reaction are tabulated for 51,580 trainees exposed to altitude in physiological training unit chambers during 1955 (table 2).

TABLE 2. *Symptoms by grade in 51,580 man chamber flights, 1955 (number per 100,000 man flights)*

Symptom	Grade of reaction			
	I	II	III	IV
Ear pain	6650	2437	514	-
Abdominal pain	2738	1187	322	12
Bends	1594	642	155	21
Sinus pain	1516	723	176	-
Toothache	285	142	118	-
Chokes	47	19	-	2
Central nervous system disturbance	12	6	6	4
Other	432	167	178	54

The incidence figures calculated for Grade IV chamber reactors show an increase from 49 per 100,000 trainees in 1954, to 85 per 100,000 trainees in 1955, and to 110 per 100,000 trainees in 1956.[10]

A "Record of Chamber Reactor" is completed on each Grade IV reactor and copies forwarded to the Surgeon General and to the School of Aviation Medicine. These records were recently reviewed for a five-year period (1950-1955) and the detailed findings reported.[11] The simple classification of these severe cases into chiefly circulatory, chiefly neurologic, or mixed is one of several attempts at categorizing these reactions.[12] Review of the 125 records, however, revealed the following classification to be of value.

CLASSIFICATION

Group 1. Persons having bends, chokes, gas, et cetera, as an initial symptom, followed by syncope or signs of impending syncope, with recovery by the time ground level was reached or within a few minutes thereafter. (Minor residual signs and symptoms such as mild pallor or sweating are not to be considered as significant provided the reactor is mentally alert and the blood pressure is normal.)

Group 2. Persons having circulatory and other autonomic signs and symptoms at altitude, who recover at ground level or during the two-hour observation period, and then have a subsequent delayed reaction.

Group 3. Persons having circulatory and other autonomic signs and symptoms at altitude which progress to immediate or delayed shock.

Group 4. Persons having syncope without preceding symptoms.

Group 5. Persons with any of numerous neurologic signs and symptoms who either proceed to recovery or have residual defect. (The pseudo-paresis seen with severe bends must be carefully excluded from this group.)

The number of reactors placed in each group is shown in table 3.

TABLE 3. *Distribution of reaction groups in 125 Grade IV chamber reactors*

Reaction group	Number	Per cent
1	65	52.0
2	4	3.2
3	4	3.2
4	5	4.0
5	47	37.6
Total	125	100.0

The signs and symptoms of pallor, sweating, nausea, and vomiting were considered to be of autonomic origin and were found associated with many of the reactions in different groups.

Changes in blood pressure and pulse were thought to represent circulatory symptoms and signs. Neurologic signs and symptoms covered a wide range and included paresis, paralysis, scotomata and other visual field losses, convulsions, inconsistent and shifting reflexes, and involuntary urination or bowel movement.

In comparing the findings on various factors analyzed, only groups 1 and 5 are used because the other three groups had so few cases.

On first appraisal of the reactor records, 22 cases were placed in group 4, but careful review of the narrative summary revealed other symptoms and signs noted prior to the loss of consciousness. The number was thus reduced to five cases in which the records showed all other signs and symptoms followed the loss of consciousness. I believe that careful observation of these five might have revealed other signs and symptoms and thus deleted group 4 in the classification.

Peak Altitude Attained and Rate of Ascent. Some reactions occurred on ascent and some on descent after peak altitude had been reached, but in any case the highest altitude attained was always recorded. Some 77 per cent of group 1 and 79 per cent of group 5 had peak altitudes of 35,000 or 40,000 feet. The rate of ascent varied between 2,000 and 4,000 feet per minute in all cases except those occurring on rapid decompression.

Time Over 30,000 Feet. Some 50 to 60 per cent of groups 1 and 5 were exposed to altitudes in excess of 30,000 feet for less than 10 minutes; 90 to 97 per cent of both groups were over 30,000 feet for less than 20 minutes (fig. 1).

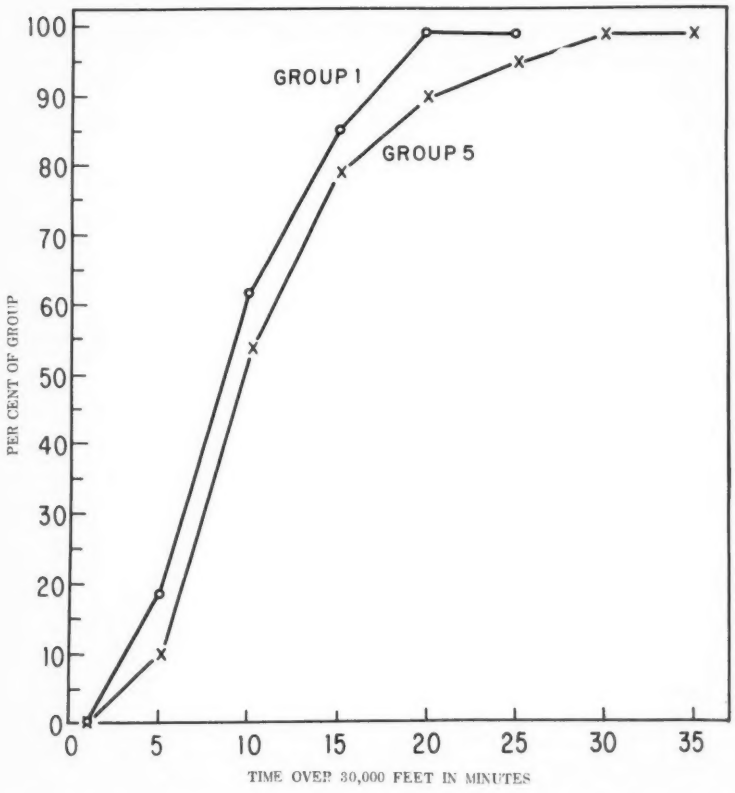

Figure 1. Per cent in reaction groups 1 and 5 who did not remain over 30,000 feet for the indicated minutes.

Body Type and Weight/Height Ratio. More than 80 per cent of the reactors were found to have a linear density (weight in pounds ÷ height in inches) in the range 2.3 to 2.7. This is not unusual as our selection process gives us personnel in the mesomorphic category. Most of the deaths in U. S. Air Force aircraft have been in overweight, nonflying personnel traveling in rear-seat passenger status.

First Symptom. Bends were the most frequently reported first symptom in all reaction groups except group 4 (the five cases

who had syncope without prior symptoms). The order of frequency for first symptoms in group 1 was bends, abdominal pain, and dizziness; while for group 5 it was bends, skin manifestations, and unconsciousness.

Signs and Symptoms in Chamber. Loss of consciousness was the most frequent symptom in groups 1, 4, and 5. Dizziness was second in group 1. In group 5, there were three other symptoms that tied for first place with unconsciousness; these were paralysis or paresis, abnormal vision, and dizziness. A high percentage of all reaction groups had bends (table 4). Chokes occurred in all groups except group 4. Abdominal distension was twice as high (33.8 per cent) in group 1 as in group 5 (19.1 per cent).

TABLE 4. *Per cent distribution of symptoms reported in the chamber in 125 Grade IV chamber reactors, 1950-1955*

Symptoms in chamber	Group				
	1	2	3	4	5
Bends	35.4	25.0	75.0		55.3
Chokes	7.7	50.0	25.0		10.6
Abdominal distension	33.8	25.0			19.1
Skin reaction	4.6	75.0			23.4
Apprehension	27.7	25.0	25.0	40.0	40.4
Pallor	16.9		25.0	40.0	6.4
Sweating	30.8	50.0	25.0		42.6
Nausea	16.9		50.0	20.0	6.4
Vomiting	16.9				19.1
Cyanosis	23.1		25.0	60.0	19.1
Dizziness	55.4	25.0	100.0	60.0	61.7
Hyperventilation	30.8	25.0	25.0	20.0	34.0
Spasm and convulsions	13.9			20.0	21.3
Headache	9.2				14.9
Abnormal vision	12.3	25.0	75.0		61.7
Paralysis or paresis					61.7
Loss of consciousness	58.5	50.0	75.0	100.0	61.7

Skin reactions occurred in only 4.6 per cent of group 1, while they occurred in 75 per cent of group 2 and 23.4 per cent of group 5. Comparisons between groups 1 and 5 are shown in figure 2.

Days in Hospital. Of the group 1 cases 87 per cent were hospitalized only two days or less. Of the group 5 cases, 49 per cent were hospitalized only two days or less, 42 per cent were hospitalized from three to seven days, and 9 per cent for 10 to 17 days.

Treatment. Only 35 per cent of group 1 reactors received treatment of oxygen and/or fluids, while 58 per cent of the 45 group 5 reactors received either oxygen, fluids, or both.

The only death in the 125 reactors was reported in detail by Adler.[12] One other death was removed from the data as it was due to explosive decompression and lung rupture. Thus some 124 reactors recovered. The questions as to the number who have a residual and the number who are still flying must remain unanswered for the present. Denitrogenation was limited to the time of the flight in most all instances.

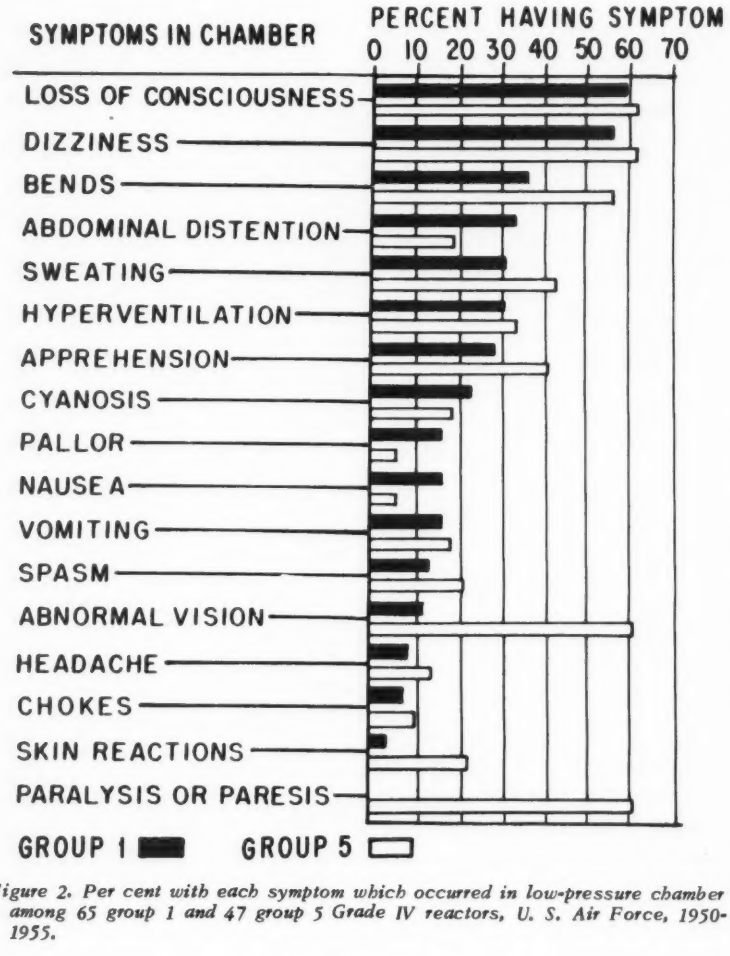

Figure 2. Per cent with each symptom which occurred in low-pressure chamber among 65 group 1 and 47 group 5 Grade IV reactors, U. S. Air Force, 1950-1955.

DISCUSSION

Loss of consciousness was the most frequently recorded symptom in three of the five reactor groups. In most instances

it was preceded by moderate to severe symptoms of dysbarism such as bends, chokes, abdominal distension, and/or skin reactions. The mechanism of this loss of consciousness remains an interesting enigma. The cause of the loss of consciousness in these cases can only be speculated on, and some form of vasovagal reaction seems a probable choice. A respiratory reflex action is possible. I believe that many if not most of the type 1 reactions are the result of vasodepressor and vasovagal reactions and thus explainable.

The basis of loss of consciousness in the neurologic reactions is unknown. Speculation as to its cause may lead to a reflex mechanism as discussed above, but further information is sorely needed. It is of interest that loss of consciousness was reported only in the in-flight reactors who died.

The reluctance to return to flying status those individuals having an episode of unexplained loss of consciousness and our inability to describe what is an acceptable stress for causing fainting (syncope) in a "normal" person, leave the disposition of such reactors as a pressing but unanswered problem.

The exposure period need not be long to produce a severe dysbarism reaction. The large percentage of reactors (90 to 97 per cent) have less than 20 minutes over 30,000 feet. This finding may be of some importance in developing safety procedures for future civilian jet airliners flying with unselected passengers.

Severe skin reactions such as rash and mottling were almost four times as frequent in the group 5 cases as in the group 1 cases. Thus skin reactions are more often associated with neurologic lesions and should be regarded as a premonitory sign of rather serious importance. A recent report by Rait[13] puts great emphasis on a fatty liver and fat emboli as the etiologic agents in these cases. Petechiae of the skin have not been reported in these cases.

The disposition of cases of decompression sickness continues to be a problem in our aviation medicine consultant centers. Although each case is evaluated individually, the huge majority of decompression sickness cases have not been returned to flying status, for in our service this implies unlimited flying status. This usual reluctance to return such cases to flying is based on the probability that such an individual is more likely to have a similar episode if again exposed to altitude. Evidence to support this probability was reported by Romano and associates.[14]

Two Air Force patients[15, 16] were returned to flying status and the outcome and their present status is unknown. The patient reported on by Rait was also returned to flying and exposed to altitude in the low-pressure chamber (37,000 feet) without recurrence of his difficulty.

It would seem that there are two questions to be answered before we can consider returning such individuals to flying status and thus exposing them to the stressful environment which produced their symptoms:

1. Does the patient have any neurologic residual?
2. Is the risk of exposing these individuals to altitudes in excess of 30,000 feet greater than that encountered in exposing persons who have never had an episode of decompression sickness?

In order to answer these questions adequately, we must gather more information. There has been no follow-up program for decompression sickness cases in the Air Force. A project is being initiated to secure the return of the 125 reactors reported in 1950-1955, and the 48 reactors reported in 1956 to this school for a follow-up evaluation. This program will include the known in-flight cases and particularly those who have been returned to flying status. It is proposed to use every means at our disposal to determine the presence of any residual defect or greater predisposition. As a step to assist in evaluation of future reactors, the "Record of Chamber Reaction" has been modified to provide more information.

Even with some increase in numbers, the total number of reactors compared to the persons trained in the U. S. Air Force is small. The British (Royal Air Force) have been concerned by the risk of such training and therefore have modified their training to render it less stressful.[7] Whittingham[17] reported a similar cessation of training for civil airline (British Overseas Airways Corporation) crews. I believe the value of the physiologic training program in preventing even more in-flight incidents of hypoxia, disorientation, dysbarism, et cetera, far outweighs any risk so far encountered.

At the present time what can we do about the treatment and prevention of neurocirculatory collapse at altitude? Due to our lack of knowledge, only prophylaxis and improved symptomatic treatment is available. On the basis of our present knowledge it would seem wise to attend to the following:

1. Indoctrinate physiological training officers and chamber observers in early recognition of symptoms and signs of dysbarism.
2. Return to ground level any person with a sign or symptom suggestive of severe dysbarism. This applies to persons in operational aircraft as well as in low-pressure chambers. Trainees with any symptom of dysbarism should not be used as hypoxia demonstrators.
3. Severe skin reactions deserve particular attention, and trainees with such reactions should be recompressed immediately. Ordinarily such cases have other symptoms or signs which would require their return to ground level.

4. Prevention of obese people from taking altitude indoctrination. These people can be eliminated by the flight surgeon on his pretraining physical examination.

5. Continue the conservative treatment policy and hospitalize all chamber and altitude reactors except those with minor bends, gas, sinus pain, earache, or toothache.

6. Consider the temporary use of Levophed (brand of *l*-norepinephrine) or Aramine (brand of metaraminol bitartrate) in the recovery room in the event the blood pressure is very low or unobtainable.

7. Initiate dextran or intravenous fluid therapy as rapidly as possible in order to have a vein open in case shock develops rapidly.

8. Use a gas mixture of 95 per cent oxygen and 5 per cent carbon dioxide for treatment of all severe reactions. Carbon dioxide has been shown to be a potent cerebral vasodilator and such action is needed in these cases.

9. Further investigation of recompression to greater than atmospheric pressure (plus 2 atmospheres) as a treatment measure as advocated by Behnke.[16] Availability of a chamber capable of doing this has been a problem in Air Force facilities, but the new School of Aviation Medicine chambers will have this capability.

10. Spinal tap should be strongly considered as a diagnostic procedure in all reactors with neurologic signs or symptoms. Some have advocated spinal puncture and removal of from 5 to 10 ml of fluid as a therapeutic measure,[1] but evidence in this area is equivocal. In practically all instances where a spinal tap was performed, the pressure and laboratory findings were normal. An exception was one of the in-flight deaths where an initial diagnosis of cerebral hemorrhage was made, bolstered by a spinal fluid pressure of 260 mm of water and the presence of 20 red blood cells per μl. In spite of the usually normal findings, improvement in the patient has been reported. However, many, if not most cases, have recovered without spinal puncture.

SUMMARY

An increased exposure of Air Force personnel to altitudes in excess of 30,000 feet has led to more reported cases of decompression sickness in both operational aircraft and low-pressure chambers. Eight Air Force in-flight (operational) reactors who survived have been reported in addition to four deaths. A plan for reporting of these cases has been initiated. During the period 1950-1955, there were 125 Grade IV dysbarism reactions reported in the Air Force. These reactions were classified into five groups which could briefly be characterized as (1) circulatory reactions following bends, et cetera, with recovery at ground level or within two hours afterward; (2) circulatory reactions with recovery and then delayed reaction; (3) circulatory

reactions progressing to shock; (4) initial syncope without previous symptoms; and (5) neurologic signs and symptoms.

Of the reactors, 90 to 97 per cent were above 30,000 feet for less than 20 minutes, and 50 to 60 per cent for less than 10 minutes. Bends was the most frequently reported first symptom. Loss of consciousness was the most frequent symptom reported during the altitude exposure. Skin reactions were most frequently associated with delayed (group 2) and neurologic (group 5) reactions. Some suggestions are made for further study of the problem and for treatment of the reactors.

REFERENCES

1. Haymaker, W.; Johnston, A. D.; and Downey, V. M.: Fatal decompression sickness during jet aircraft flight; clinicopathological study of 2 cases. *J. Aviation Med.* 27: 2-17, Feb. 1956.

2. Stembridge, V.: Personal communication.

3. Fryer, D. I. (RAF): Personal communication.

4. Fulton, J. F. (editor): *Decompression Sickness: Caisson Sickness, Diver's and Flier's Bends and Related Syndromes.* Compiled under auspices of Subcommittee on Decompression Sickness, Committee on Aviation Medicine, Division of Medical Sciences, National Research Council, Washington, D. C. W. B. Saunders & Co., Philadelphia, Pa., 1951.

5. Le Fritz, N.: Physiological Training Officers Notes, Smoky Hill Air Force Base, Kans., Dec. 1955.

6. Mebane, J. C.: Clinical neuropsychiatry in aviation medicine; survey of 100 military problem cases. *J. Aviation Med.* 26: 471-478, Dec. 1955.

7. Döbeln, W. Von, and Höök, O.: Olycksfall vid undertryckskamnarproo. *Medd. flyg. nav. med.* No. 4, pp. 14-16, 1954.

8. Explosive decompression experience in U. S. Air Force. Compiled from reports to the Directorate of Flight Safety, Norton Air Force Base, Calif.

9. U. S. Air Force Regulations: AFR 50-27, Physiological Training Program.

10. Ames, S: Personal communication.

11. Berry, C. A., and Wayne, H. H.: Grade IV chamber reactions or neurocirculatory collapse occurring in the USAF 1950-1955. Presented at Aero Medical Association Meeting, Denver, Colo., May 1957. (abstract) *J. Aviation Med.* 28: 192, Apr. 1957.

12. Adler, H. F.: Neurocirculatory Collapse at Altitude, Special Project, U. S. Air Force, School of Aviation Medicine, Randolph Air Force Base, Tex., June 1950.

13. Rait, W. L.: The aetiology of post decompression shock. Presented at Aero Medical Association Meeting, Denver, Colo., May 1957.

14. Romano, J., and others: Syncopal reactions during simulated exposure to high altitude in decompression chamber. *War Med.* 4: 475-489, Nov. 1943.

15. Halbouty, M. R., and Long, D. R.: Neurocirculatory collapse in aircraft flight; report of case. *J. Aviation Med.* 24: 301-307, Aug. 1953.

16. Schneck, S. A.: Decompression sickness at medium altitude. *U. S. Armed Forces M. J.* 8: 1366-1370, Sept. 1957.

17. Whittingham, H. E.: Aero-medical problems of jet passenger aircraft. *J. Aviation Med.* 25: 440-450, Oct. 1954.

18. Behnke, A. R.: Decompression sickness. *Mil. Med.* 117: 257-271, Sept. 1955.

LIMITED THORACOTOMY AND ENZYMATIC THERAPY IN THE TREATMENT OF EARLY CLOTTED HEMOTHORAX

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THE medical literature on treatment of the patient with hemothorax is legion. In the days before the development of thoracic surgery, a clotted hemothorax committed a patient to the life of a pulmonary cripple with all the future complications of a fibrothorax. Surgeons have learned much from experience gained in World War II about the treatment of this condition so that at the present time therapy has become fairly well standardized.

Ideally, the treatment of hemothorax from any cause is the early, continued, and complete removal of blood and air from the pleural cavity. Whether this is accomplished by needle aspiration, intercostal catheter drainage, or, rarely, thoracotomy if indicated, is a matter of conjecture and depends on the preference of the surgeon. However, there will be patients where the most diligent pursuit of this objective fails and who remain with a clotted hemothorax. It is this type of patient—the one with an early clotted hemothorax—with which this article is concerned.

Enzymatic therapy (streptokinase and streptodornase) in hemothorax and empyema has been advocated by Sherry, Tillett, and Read.[1] Others have reported encouraging results with these enzymes.[2-5] On the other hand, some question the use of enzymatic therapy. It is our belief that when enzymatic therapy is applied to the selected case, *i. e.*, an *early* clotted hemothorax, that much benefit may be gained in conjunction with other forms of therapy.

Decortication of an encapsulated lung due to clotted hemothorax or organized empyema is now an accepted surgical procedure. Much is owed to Samson and Burford[6] and Tuttle, Langston, and Crowley[7] for their work in developing this procedure during and after World War II. The optimum time to perform this procedure has been stated to be about three weeks after the

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occurrence of the hemothorax and, indeed, if performed at this time, is a simple and safe procedure. In the borderline case, however, where there is a question of whether decortication is really indicated or not, too often the decision is to "wait two or three months and further evaluate the patient." In military life as well as civilian, the implications of such a decision are clear. In either case the result is no gainful work during the observation period or, at best, work of a limited nature.

Within 10 days after the onset of a hemothorax it usually will become apparent to the attending physician whether or not he will be successful in evacuating all blood from the pleural cavity. The number of chest aspirations required, the success of such aspirations, clotting tendency of the blood removed, radiographic appearance of the chest, examination of the patient, et cetera, will aid in arriving at an early decision. Once it has been determined that further aspiration of the chest, whether by needle or intercostal catheter drainage, is futile, and if any significant degree of hemothorax remains, we have adopted the following plan of therapy: A "limited" thoractomy is performed, resecting about 4 to 5 inches of the 6th or 7th rib laterally, with the patient in the standard thoracotomy position. The pleural cavity is opened and all soft clots are removed. Pockets of encapsulated blood are broken up, and fluid is removed by suction. At this stage of the disease any fibrinous deposits on the lung are very loose and lend themselves to easy removal. A lighted retractor within the chest cavity is very helpful and provides adequate illumination. No effort is made to do a complete decortication—only the clots and fluid are removed, pockets broken up, and loose filmy exudates over the lung surfaces are removed. A large, right-angled, No. 45 French intercostal catheter is then inserted in the lowermost portion of the chest in the midaxillary line with the intrapleural end of the tube pointing posteriorly. This is then connected to a water trap.

Immediately following the operation, 2 ampules of Varidase (brand of streptokinase-streptodornase) totaling 200,000 units of streptokinase and 50,000 units of streptodornase are mixed in 20 ml of physiologic saline solution and inserted into the pleural cavity through the chest tube, which has been clamped. Instructions are given the nurse to leave the tube clamped for 12 hours and then to release it. If during the 12-hour period, the patient's temperature rises to 104°F, the tube is immediately unclamped. This procedure can be repeated daily if indicated by lack of clearing on the daily chest roentgenogram. Usually only two injections of enzymes are required.

Although our experience with "limited" thoracotomy, "limited" decortication, and subsequent enzymatic debridement of the pleural cavity covers only a few cases, the results achieved have been excellent and, we believe, warrant reporting.

CASE REPORTS

Case 1. A 26-year-old Caucasian in March 1957 suffered a right spontaneous pneumothorax for which he was successfully treated at another hospital. On 25 July he developed a spontaneous right hemothorax, was admitted to another hospital, and then transferred to this hospital two days later. Needle aspiration of the right side of the chest on the day of admission resulted in removal of 350 ml of blood. A roentgenogram showed very little change following aspiration. The following day, 225 ml of thick blood, which clotted in the needle and syringe, were removed. Aspirations were performed daily thereafter without success (fig. 1). On 2 August 1957, eight days after the onset of the spontaneous hemothorax, a "limited" right thoracotomy was performed, removing about 5 inches of the midportion of the 6th rib.

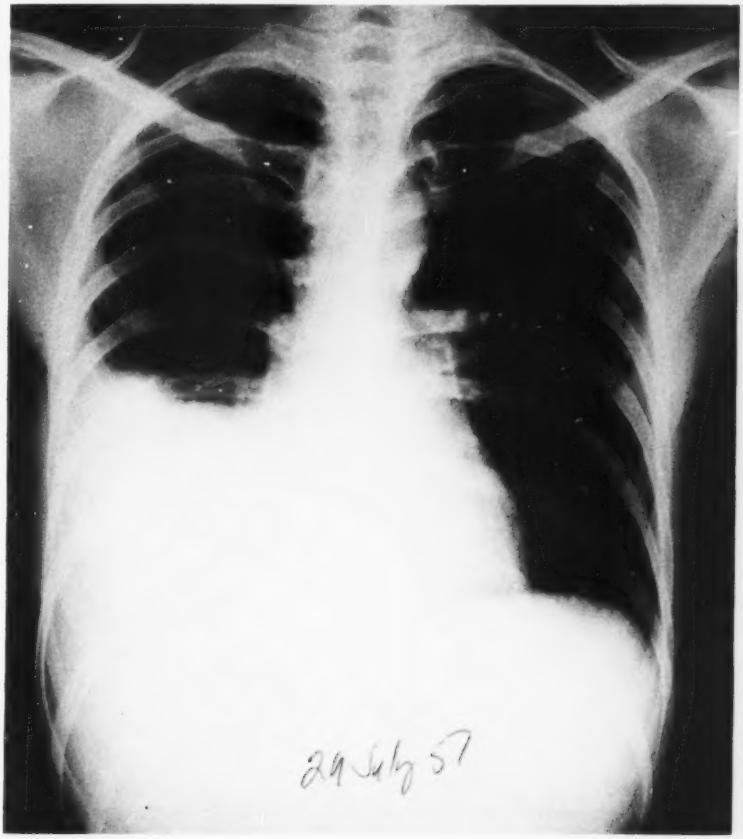

Figure 1 (case 1). Preoperative roentgenogram of chest showing clotted hemothorax.

All clots were removed, fluid was aspirated by suction, and some loose fibrinous exudate was removed from the lung surfaces. A No. 45 French, right-angled, chest catheter was placed in the 9th interspace in the midaxillary line and connected to a water trap. That afternoon 200,000 units of streptokinase and 50,000 units of streptodornase were placed in the pleural cavity through the chest tube and the tube clamped. The patient was then turned every half hour to ensure adequate distribution of the enzymes within the chest. Two hours following this the patient's temperature rose to 102.6°F, the highest attained. Twelve hours after instillation, the chest tube was unclamped and about 1,000 ml of bloody serosanguineous material collected in the water trap. This procedure was repeated the following day and upon releasing the clamp from the chest tube, only 100 ml

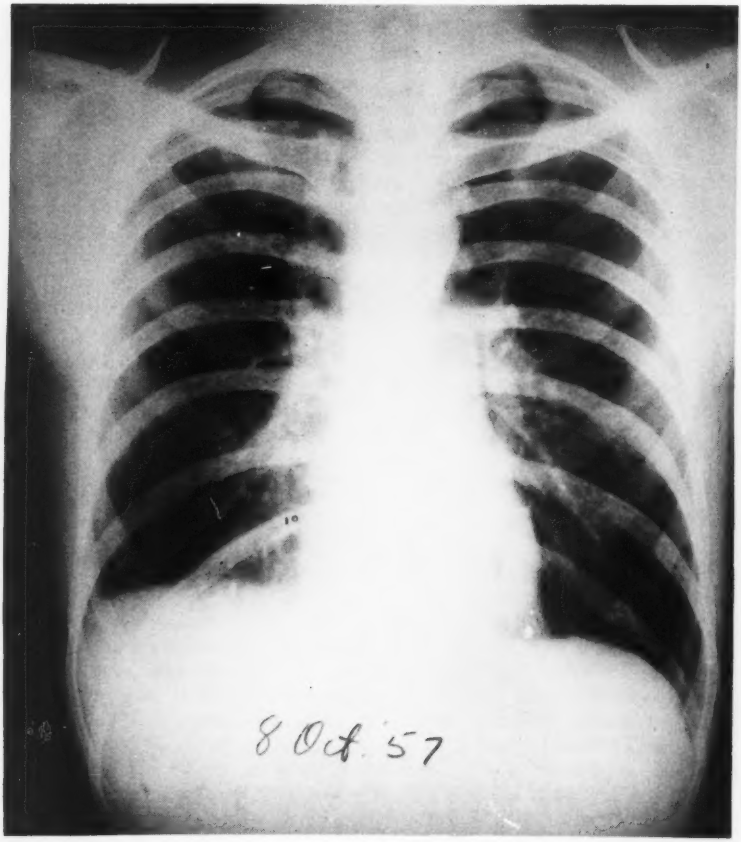

Figure 2 (case 1). This roentgenogram demonstrates the end result two months after "limited" thoracotomy and enzyme therapy.

of fluid drained into the bottle. The chest tube was removed the following day and convalescence thereafter was uneventful. The patient was sent on convalescent leave 11 days following thoracotomy. A follow-up roentgenogram (fig. 2) on 8 October revealed very satisfactory resolution of the hemothorax and complete lung expansion.

Case 2. A 26-year-old man was admitted to this hospital on 13 May 1957, three hours after an automobile accident in which he suffered multiple abrasions and contusions and a fracture of the 10th rib on the left plus a compression fracture of the 10th dorsal vertebra. The admission chest film showed a minimal left hemopneumothorax. Follow-up roentgenograms on 23 May 1957 showed a delayed massive hemothorax. A tap done at this time produced 1,550 ml of dark-red blood,

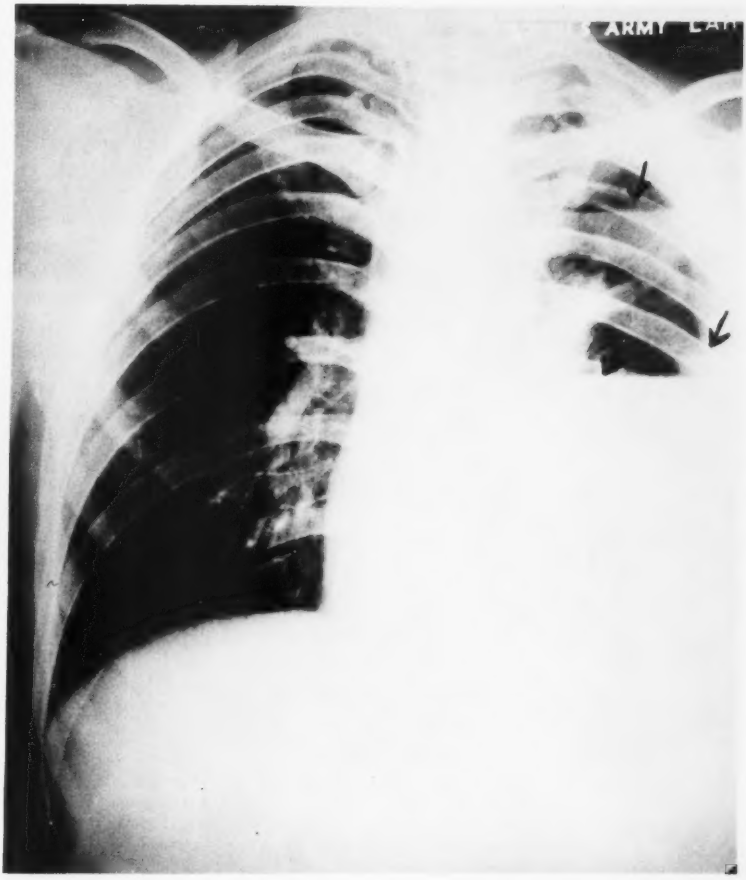

Figure 3 (case 2). Preoperative roentgenogram showing multiple fluid levels.

which clotted immediately on standing. A repeat tap the next day produced only 440 ml of blood, which also clotted immediately on standing. Further taps during the next two days produced little if any blood and it was believed that the patient had a clotted hemothorax (fig. 3). A limited thoracotomy was performed on 27 May. At this time the lung was noted to be encased in a semifibrous peel and could not be fully expanded. Multiple pockets of clotted and unclotted blood were removed by breaking up the loculations and aspirating the blood and debris (1,225 ml). A No. 45 French chest tube was placed in the chest and connected to a water trap. The next day 200,000 units of streptokinase and 50,000 units of streptodornase were injected through the thoracotomy tube into the chest. The tube was clamped and opened

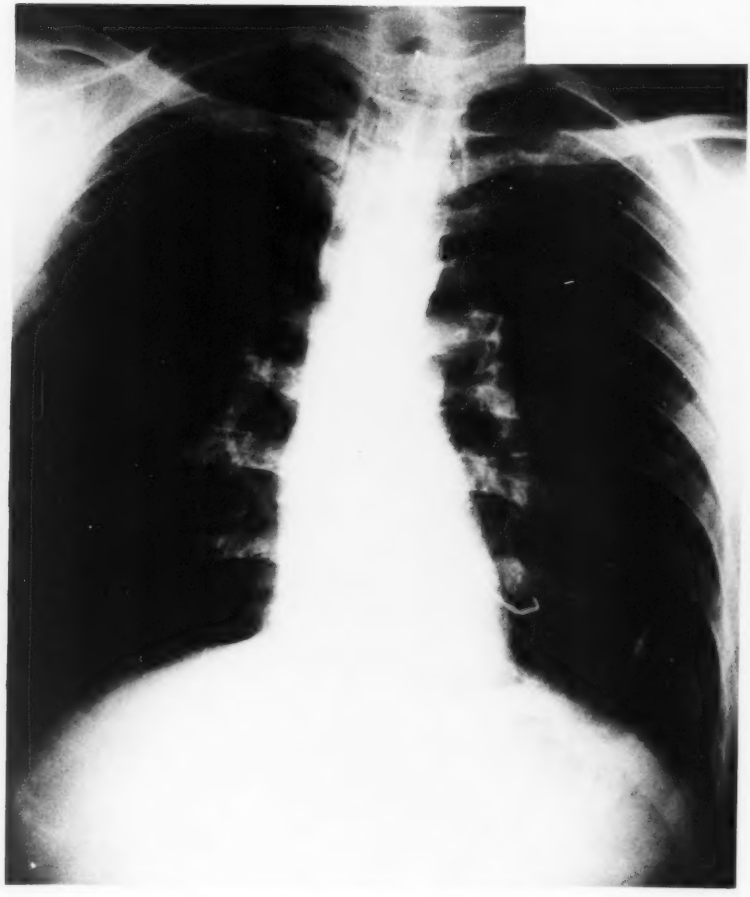

Figure 4 (case 2). Postoperative film.

after 12 hours. The highest temperature elevation was 102°F. Upon unclamping the tube, 1,500 ml of bloody serous fluid was expelled into the water-seal drainage bottle. The next day another similar dose of the enzymes was injected and when unclamped after 12 hours, 1,000 ml of fluid was obtained. The tube was removed the following day and from then on the patient made an uneventful recovery (fig. 4).

DISCUSSION

There will be some surgeons who upon reading this paper will raise the question of "why a limited thoracotomy?" "Why not perform a standard thoracotomy and finish the job then and there?" The answer to these questions is that if some type of operation is to be performed, a "limited" thoracotomy has the advantage of less operative trauma, less reluctance on the part of the surgeon to operate in a borderline case, and fewer postoperative complications such as chest pain, failure to cough, and atelectasis. Convalescence is rapid and uneventful. Hospital stay is definitely shorter following "limited" thoracotomy.

It is emphasized again that the regimen outlined here is for a very select group indeed, and is not advocated for late cases of clotted hemothorax or organized fibrothorax or empyema. If it is ascertained at "limited" thoracotomy that a more formal operation is indicated by the findings within the chest (thick, dense, adherent exudate over the lungs, diaphragm, mediastinal pleura, et cetera) it is a simple matter to extend the incision to that of a standard posterolateral thoracotomy size and carry out a formal decortication.

SUMMARY AND CONCLUSIONS

"Limited" thoracotomy and "limited" decortication in conjunction with enzyme therapy is proposed as another form of treatment in *early* clotted hemothorax (8-12 days after onset). Indications that open thoracotomy will be required for clotted hemothorax usually become manifest 8-10 days after the onset of hemothorax. A plea for earlier operation is made. Early operative intervention results in a technically easier procedure with resulting better lung function for the patient. Early "limited" thoracotomy and decortication along with enzyme therapy has achieved excellent results in a small series of patients, has resulted in fewer days in the hospital, and a much lower morbidity rate.

REFERENCES

1. Sherry, S.; Tillett, W. S.; and Read, C. T.: Use of streptokinase-streptodornase in treatment of hemothorax. *J. Thoracic Surg.* 20: 393-417, Sept. 1950.

2. McCroskey, C. E., and Hardin, C. A.: Tryptic débridement of clotted hemothorax; experimental and clinical study. *A. M. A. Arch. Surg.* 66: 650-655, May 1953.

3. Read, C. T., and Berry, F. B.: Utilization of streptokinase-streptodornase in patient with hemopneumothorax and patient with postpneumonectomy sanguineous coagulum. *J. Thoracic Surg.* 20: 384-392, Sept. 1950.

4. Streete, B. G., and Thomas, D. E.: Streptokinase and streptodornase-in thoracic surgery for pulmonary tuberculosis. *J. Thoracic Surg.* 34: 49-52, July 1957.

5. Tillett, W. S.; Sherry, S.; and Read, C. T.: Use of streptokinase-streptodornase in treatment of postpneumonic empyema. *J. Thoracic Surg.* 21: 275-297, Mar. 1951.

6. Samson, P. C., and Burford, T. H.: Total pulmonary decortication; its evolution and present concepts of indications and operative technique. *J. Thoracic Surg.* 16: 127-145, Apr. 1947.

7. Tuttle, W. M.; Langston, H. T.; and Crowley, R. T.: Treatment of organizing hemothorax by pulmonary decortication. *J. Thoracic Surg.* 16: 117-126, Apr. 1947.

THE PHYSICIAN'S MOST PRECIOUS POSSESSION

"Ordinarily, nowadays, most patients can be told their diagnosis as soon as it is made. If it is cancer, there is probably therapy available to cure it, or at least to control it for a while. Radical surgery and radical irradiation are so severe—give the patient so much discomfort, risk his life so much, and carry so much in the way of bothersome sequelae—that one cannot expect an intelligent patient to accept them without proper reason. But the diagnosis should not be given until it is certain, or at least as certain as one can make it. A doctor needs a vivid appreciation of the emotional turmoil that he inflicts on his patient with a diagnosis of cancer. If the diagnosis is withdrawn, the sense of relief is lovely, but it does not recompense the patient for what, in his mind, he has already suffered. If the doctor tells his patient that the tumor is benign and then later has to explain that it is cancer in order to get him to submit to the necessary operation, the doctor can lose the patient's confidence. His trust in his physician is one of the patient's most precious possessions. I am not talking about the fact that it is the physician's most precious possession, too, or that it is amazing how so many ordinary persons can have such trust grow up between them. What I wish to emphasize is that this trust must not be compromised."

—R. R. NEWELL, M. D.
in *Journal of Chronic Diseases*
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PRIMARY SPLIT-SKIN GRAFTING FOR TREATMENT OF LARGE PILONIDAL CYSTS

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A PROBLEM of considerable importance in the military service is the treatment of pilonidal disease. Buie and Curtiss[1] stated that 78,924 patients with this disease were admitted to Army hospitals between 1941 and 1945. Similarly, in the Navy 630,701 man-days were lost and 7,409 operations were performed between 1944 and 1951 because of pilonidal disease. The high incidence in the younger age group is well established.

The length of time necessary for healing varies according to the type population being treated. In military hospitals, it necessarily is prolonged because the patients usually are retained on the "sick list" until they are fit for full duty; in most instances this means until the wound is completely healed. Experience has shown that if these men are returned to duty before complete healing takes place, additional man-days are lost because of the higher incidence of recurrence.

The reported "healing time" for the different operative procedures varies according to the prejudices of the authors and their particular technic. Swinton and Markee[2] reported a healing time of 3 to 8 weeks in a group of 6,625 cases where only excision of the pilonidal cyst was done. Berkowitz[3] listed the average healing time required for various methods of treatment as follows: open packing, 86 days; partial closure, 55.2 days; and primary closure, 30.6 days.

Weeks and Young[4] reported an average hospitalization of 52 days with the open method of treatment and 33 days using the closed method. Palumbo, Larimore, and Katz,[5] reporting on 171 cases, gave the average period of hospitalization for the open method as 17.2 days, for primary closure as 22.4 days, and for marsupialization as 16.9 days. No mention or reference was made to the actual healing time or days lost from work.

An additional factor in this disease, which accounts for the excessive number of man-days lost, is the high incidence of

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recurrence. Probably, as indicated by Swinton and Markee, one of the most frequent causes of recurrence is improper aftercare of the wound. Turell stated: "The tendency to recurrence following any operation characterizes this lesion. It is now almost universally realized that recurrence following an operative procedure is seldom due to overlooked remaining pilonidal sinuses, but is often the result of the incomplete obliteration of dead space which has become infected."[6]

The high recurrence rate following primary closure of a pilonidal cyst is well established. Palumbo, Larimore, and Katz reported an over-all recurrence rate of 21.9 per cent in a series of 1,165 cases reviewed in the literature, where primary closure was used. The incidence of recurrence is high with other procedures as well. Swinton and Markee, reporting on the results with 123 patients treated between 1946 and 1952 at the Lahey Clinic, Boston, Mass., showed an 18 per cent recurrence rate with excision only, 6.6 per cent with marsupialization, 32 per cent with excision and simple primary closure, and 19 per cent with excision and primary closure using a modified Pope technic. Kleiman[7] had an over-all recurrence rate of 16.2 per cent in a group of 481 patients treated by various methods. In another group of 118 patients reported by Kleitsch and Cherry,[8] there was a 9.3 per cent recurrence rate. Dwight and Maloy[9] reported an average recurrence rate of 11 per cent in 411 cases followed for a year or more. As shown in table 1, the rate varied from 6 to 18 per cent depending on the operative procedure employed.

TABLE 1. *Per cent of recurrence in patients followed for one year or more after various operations for pilonidal sinus.*[9]

Type of Operation	Cases (number)	Recurrence (per cent)
Excision and simple closure	24	8
Excision and partial closure	17	6
Excision and gluteal-flap closure	159	18
Excision and wire closure	48	12
Excision and packing	75	12
Incision and packing (abscesses)	40	7
Incision and packing	48	12

In an attempt to improve on these results, I employed a method that had the advantages both of wide excision, as used in the block excision, and of primary closure. This was accomplished by use of a primary split-graft applied to the wound after a block excision had been performed. Neither the method nor the basic technic are new or original, and although only a few cases treated in this way appear in the literature,[4, 10–12] it is probable

that this method has been long used but little reported.[13] Boger and Pinkham[12] presented the largest series, a group of 25 patients. They had an average postoperative hospital stay of 28.2 days, the longest stay being that of a patient in whom an additional cyst was found later and was not treated by skin graft. The recurrence rate in this series was not given.

METHOD AND TECHNIC

The technic used is simple and effective as long as one adheres to sound surgical principles. Grafts are applied only to tissue free of inflammatory changes. To ensure this, the patients are placed on sitz baths and antibiotics several days prior to operation. If the cyst is not draining freely or is fluctuant, the sinus tract is opened by sharp incision and packed open with iodoform gauze for 24 hours. The patients are placed on a liquid diet 24 hours prior to surgery, and the lower bowel is thoroughly cleansed with repeated tap-water enemas.

The operation is performed under spinal anesthesia. To prevent contamination of the donor site, the split graft is taken prior to probing and excision of the pilonidal cyst. The posterior surface of either the right or left thigh is used. The graft is taken with a Barker Vacuotome* (fig. 1). The donor site is covered with fine mesh silk and a sterile pressure dressing applied. Care is taken to apply pressure from the toes to above the upper limits of the donor site to prevent edema distal to the dressing.

Following application of the dressing, the sinus tracts are probed circumferentially to determine the approximate size of the cyst and its ramifications (fig. 2). A wide excision of the cyst or sinus tracts down to the sacrococcygeal fascia is done, taking care not to enter the cyst or sinus cavity at any point. Particular care is taken to secure hemostasis.

The graft is applied to the pilonidal wound, anchoring it with a continuous No. 00000 plain catgut suture running in a vertical manner in the midline and extending parallel rows of suture material at 1 cm intervals, from the midline. The graft is anchored circumferentially to the wound edges with a continuous locked suture of the same material. A Stent type of dressing using a foam rubber form is anchored in place with circumferentially placed No. 2-0 black silk sutures. Additional relaxation of the grafted area is obtained by placing a pressure dressing over the Stent dressing, using fluffed gauze and long adhesive strips.

Postoperatively the patient is placed on antibiotics and bed rest for five days. He is given a liquid diet supplemented with a high vitamin intake, especially ascorbic acid. Bowel movements are discouraged by the use of paregoric. On the first post-

*A type of dermatome developed by Dr. Donald E. Barker in 1948.

operative day the supplemental pressure dressing is removed and the Stent dressing inspected. A cradle is placed over the bed and the grafted site is exposed. On the fifth postoperative day the Stent dressing and all nonabsorbable sutures are removed. A thorough débridement of all nonviable tissue is done and a sterile pressure dressing reapplied.

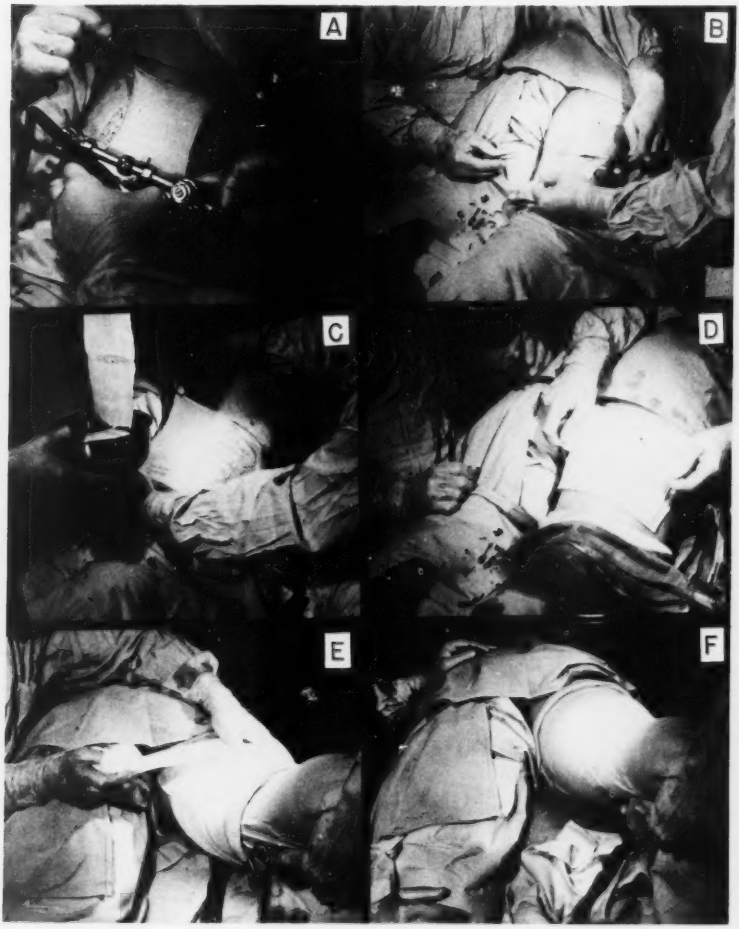

Figure 1. Method of obtaining split-skin graft and applying pressure dressing to the donor site.

The patient is placed on a regular diet and the frequency of changing dressings is governed by the number of bowel movements thereafter and the degree of soilage of the grafted area.

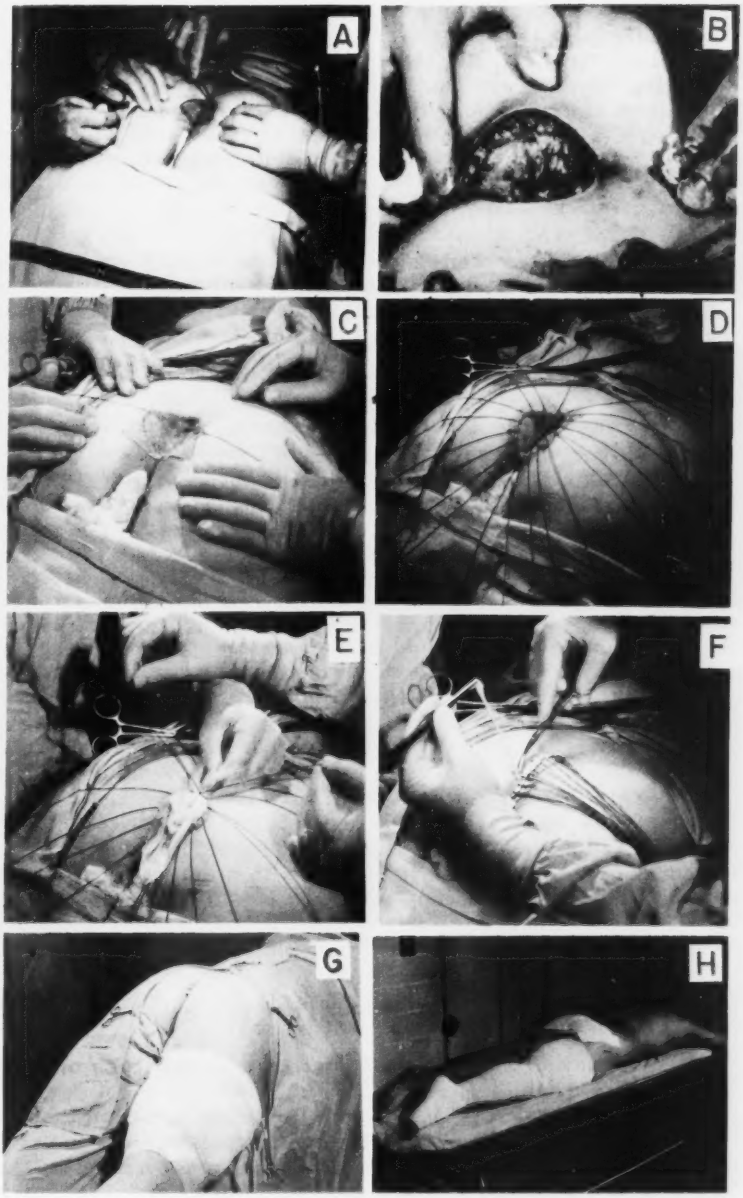

Figure 2. Method of excising the cyst and applying the graft and Stent dressing.

A minimum of two changes of dressings per day is required. We have found that the use of sitz baths has not been detrimental to grafted skin that has taken, if started after the sixth postoperative day. We use them earlier if there are any signs of infection or inflammation. The donor-site dressing is removed between the 10th and 14th postoperative days, and the patient is returned to duty as soon as the wound has completely epithelized.

RESULTS

In an attempt to correlate the results of this method with those of other methods, two groups of patients were selected at random during the period of 1950 to 1956. One group of 50 patients were treated by marsupialization or block excision and a second group of 49 were treated by block excision and primary split-skin graft.

In the first group the average number of hospital days necessary for complete healing was 66.14. The average postoperative hospital period was 59.34 days. No attempt was made to determine the recurrence rate in this group because this figure has been previously documented by many other writers. In the second group the average number of hospital days necessary for complete healing was 32.65. The average postoperative period for this group was 25.82 days.

Of the 49 patients treated in this manner, 39 were followed, either by physical examination or questionnaire, for from 6 months to 5 years. Of these 39 patients, 5 complained of slight drainage at the operative site requiring follow-up treatment on an outpatient status. Only 1 of the 39 (2.6 per cent) required further hospitalization. He had a block excision of a pilonidal cyst and a primary split-skin graft on 27 May 1955. The postoperative healing time was 19 days, and he required a total of 26 days' hospitalization before he could be returned to full duty. He returned to the hospital in 6 months with a diagnosis of ischioanal abscess. Proctoscopy did not reveal an internal sinus tract. The abscess was incised and drained. When inflammation subsided, probing of the cavity revealed a tract that ran up to the lower border of the previously grafted site. In this case recurrence resulted from incomplete excision of the pilonidal sinus at the time of the original procedure.

It is believed that the postoperative convalescence and hospitalization have been materially reduced using this method, and follow-up studies indicate a lower recurrence rate. The procedure has its disadvantages in that the preoperative preparation, the operation, and postoperative care are more time consuming than they ordinarily are in block excision or marsupialization. Other disadvantages pointed out by previous writers have not been borne out in this series. Turell stated

that grafted skin in the sacrococcygeal area cannot withstand the strain incident to full military physical duty status or corresponding stress and strain in civilian pursuits. He believed that grafted skin is easily affected by slight trauma.

In this series all patients with split grafts were sent back to full duty at the average time indicated (25.82 days). Except for the one recurrence, there has been no indication over the past five years that the grafted areas have not withstood the rigors of active military duty. Indeed, one of the patients, following the split-graft procedure, did duty in Korea during the Korean Conflict and was awarded the Legion of Merit for outstanding services. He has been followed for five years without a recurrence and has no complaints referable to either the donor or recipient sites.

This series also bears out an observation made by Hubly, Mustard, and Stiefel[11] that, although the graft is sutured directly down upon the sacrococcygeal ligament, no patient has complained of pain or discomfort in that area. Another criticism of this method has been that grafts applied to fresh wounds leave large defects. This has not been a problem because, as Hubly and associates pointed out, the original operative defect is lessened by some 30 per cent to 50 per cent as a result of contracture. Furthermore, experience in this series indicates that the greater the tension placed on the graft at the time of its application, the greater will be the degree of subsequent contraction.

Some authors[10] propose a delayed skin-grafting procedure, applying the graft after clean granulations have formed. It is thought that such a delay is not necessary if strict attention is paid to sterile precautions. There has been no difficulty with irregularly surfaced or cosmetically unsatisfactory grafts. An additional one to two weeks of hospitalization is added if one waits for clean granulations to appear before grafting.

Thus far there has been no cause for regret in employing the primary split-graft procedure in the treatment of pilonidal disease. The results are encouraging enough to allow this procedure to be employed as one of the routine methods for treating large pilonidal cysts where convalescence would be unduly prolonged with one of the accepted procedures for the "open method" of treatment.

SUMMARY

The prolonged hospitalization and convalescence usually necessary for the treatment of pilonidal disease are of particular concern to the armed services. To meet this problem, a method of treatment by block excision followed by primary skin-grafting is proposed.

The encouraging results obtained in 49 cases suggest that this method is advantageous for the treatment of large pilonidal cysts where convalescence would be unduly prolonged with one of the accepted procedures used in the "open method" of treatment.

REFERENCES

1. Buie, L. A., and Curtiss, R. K.: Pilonidal disease. *S. Clin. North America*, 32: 1247-1259, Aug. 1952.

2. Swinton, N. W., and Markee, R. K.: Present status of treatment of pilonidal sinus disease. *Am. J. Surg.* 86: 562-571, Nov. 1953.

3. Berkowitz, J.: Sacrococcygeal pilonidal cyst. *Am. J. Surg.* 77: 477-490, Apr. 1949.

4. Weeks, R. B., and Young, G. G.: Sacrococcygeal cysts; report of 200 cases in Army hospital. *Am. J. Surg.* 60: 260-263, May 1943.

5. Palumbo, L. T.; Larimore, O. M.; and Katz, I. A.: Pilonidal cysts and sinuses; statistical review. *A. M. A. Arch. Surg.* 63: 852-857, Dec. 1951.

6. Turell, R.: Pilonidal (sacrococcygeal) cyst and sinus; evaluation of some of the recently proposed therapeutic measures. *New York State J. Med.* 47: 977-980, May 1, 1947.

7. Kleiman, A.: Pilonidal cyst; follow-up studies on 481 patients. *U. S. Armed Forces M. J.* 3: 575-580, Apr. 1952.

8. Kleitsch, W. P., and Cherry, L. D.: Pilonidal sinus—rational scheme of treatment. *Mil. Surgeon* 111: 96-106, Aug. 1952.

9. Dwight, R. W., and Maloy, J. K.: Pilonidal sinus; experience with 449 cases. *New England J. Med.* 249: 926-930, Dec. 3, 1953.

10. Williams, A. C.: Skin grafting in treatment of extensive pilonidal disease and anal fistula. *Am. Surgeons* 17: 1137-1144, Dec. 1951.

11. Hubly, J. W.; Mustard, R. L.; and Stiefel, R. A.: Skin grafting for recurrent pilonidal cyst. *J. Michigan M. Soc.* 46: 921-923, Aug. 1947.

12. Boger, E. V., and Pinkham, E. W., Jr.: Primary split-skin graft in treatment of pilonidal cysts. *U. S. Armed Forces M. J.* 2: 1733-1736, Nov. 1951.

13. Connelly, J.: Personal communication.

A SHIGELLA-LIKE INTERMEDIATE SEROTYPE: ALKALESCENS-DISPAR 01 var. KOJI

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DURING the course of a dysentery epidemic among impounded prisoners of war in the summer of 1951,[1] several thousand cultures were submitted to this laboratory by the Dysentery Research Unit. The majority of these were members of the genus *Shigella,* notably of the Flexner group, and occasionally *Salmonella* sp.[2,3] On routine examination in this laboratory, a group of apparently related, aberrant strains was encountered and subjected to further study.

It was found that these organisms were capable of producing only acid when cultured in broth containing glucose, trehalose, maltose, arabinose, xylose, or sorbitol. Those which were not fermented after 15 days' incubation were sucrose, lactose, raffinose, rhamnose, mannitol, dulcitol, inositol, dextrin, glycerol, adonitol, and salicin. The organisms were nonmotile, formed indole, did not produce H_2S, and were unable to utilize citrate (Simmons Citrate Agar) as a sole source of carbon. The test for trimethylamine oxide reduction[4] was negative; the methyl red test was positive; acetylmethylcarbinol was not produced; and nitrate was reduced. The organisms grew at 45.5°C.[5] The KCN and ninhydrin tests were negative, and urea was not split.

These reactions are consistent with those expected of members of the genus *Shigella;* however, in the slide agglutination tests using cell suspensions[6] there were no reactions. Therefore, as species of *Shigella* may possess thermolabile blocking substances, the suspensions were heated to boiling. The heated antigens reacted with the Flexner group serum, but with none of the specific sera of this group. Alkalescens-Dispar 01 (A-D 01) serum caused marked agglutination by slide test, and slight reactions in serum against *Shigella dysenteriae* 1 were observed.

Although these organisms reacted in both *S. dysenteriae* 1 antiserum and in A-D 01 serum, the biochemical reactions are consistent with neither of those species—xylose was fermented, indole was formed, and the organisms grew at 45.5°C, of which

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the Shiga's bacillus is incapable but which is characteristic of A-D 01. On the other hand, mannitol was not fermented, trimethylamine oxide was not reduced, the KCN and ninhydrin tests were negative, which is inconsistent with A-D 01 but not with *S. dysenteriae* 1.

Thus, this group of organisms, of which strains 891, 2499, and 3162 were representative, did not correspond in its antigenic structure and biochemical reactions to any of the known members of the genus *Shigella*. It became apparent that there should be further investigation with respect to the antigenic make-up of the group.

MATERIALS AND METHODS

Antigens. The antigens used for the production of antiserum were prepared by inoculating tryptose phosphate agar slants and incubating at 37°C for 24 hours. The growth was suspended in physiologic saline solution containing 0.3 per cent formalin, and filtered through glass wool. The filtrate was held at 37°C overnight and centrifuged; the cells were washed once in formalinized saline and resuspended. The final suspension was adjusted to a density corresponding to the No. 3 McFarland nephelometer (about 9×10^8 organisms/ml) and tested for sterility before use. These antigens were used without further treatment as unheated antigen, or were boiled for 5 minutes and used as heated antigen.

The antigens for use in the absorptions were prepared from cells grown on tryptose phosphate agar in culture bottles. These were washed from the surface with saline, centrifuged, and used with no further treatment in the case of the unheated antigen. The heated antigen was boiled for 5 minutes before the suspension was centrifuged.

The antigens used in the tube agglutination tests were prepared similarly to those used for the absorptions, except that the suspensions were adjusted to the No. 3 McFarland nephelometer and stored in the refrigerator at 5°C until used.

Antisera. The antisera were obtained from 2.5 to 3.0 kg rabbits following serial, intravenous inoculations. The rabbits were exsanguinated by intracardiac puncture after trial bleeding showed the antiserum to be of sufficiently high titer. The blood was collected in sterile bottles, allowed to coagulate at room temperature, and stored at 5°C overnight. The serum was harvested by pipet, preserved with Merthiolate (brand of thimerosal) (1: 10,000) and stored at 5°C.

Absorption. The absorptions were performed by slurrying the desired amount of diluted serum (1:10) with the proper absorbing antigen in equal parts. The mixture was incubated at 45°C for two hours with periodic agitation. This suspension was centrifuged to remove the cells, and the serum tested for activity against

the absorbing antigen by the slide agglutination test. This procedure was repeated until all unwanted antibody had been removed.

Agglutination tests. The slide agglutination test was performed according to the technic described by Ewing.[6]

The tube agglutinations were accomplished by preparing two-fold dilutions of the serum and adding an equal volume of the antigen. After thorough mixing, the tubes were placed in a 37°C water bath overnight. Titers include the further two-fold dilution by the antigen.

RESULTS

The agglutination titers of the six sera were determined against heated and unheated homologous antigens and against the two related heterologous types, as well as *Shigella dysenteriae* 1 and Alkalescens-Dispar 01. These titers are presented in table 1. The unheated 891 antigen has an active heat-labile blocking substance which severely inhibits the antigen-antibody reaction in the homologous and heterologous sera. This phenomenon is hardly observed with strain 2499 and slightly so with strain 3162. The antigen related to *S. dysenteriae* 1 appears to be altered by heating, as this agglutinin is reduced in quantity in sera against the heated 891 type antigens. This is a demonstration of a K antigen associated with the 891 type and *S. dysenteriae* 1. Such treatment, on the other hand, has little effect on the titers against A-D 01 antigen.

TABLE 1. *Serologic cross-reaction of strains 891, 2499, and 3162 in terms of reciprocal of highest positive dilution*

Serum	Antigen									
	891 unheated	891 heated	2499 unheated	2499 heated	3162 unheated	3162 heated	S. dysenteriae 1 unheated	S. dysenteriae 1 heated	Alkalescens-Dispar 1, unheated	Alkalescens-Dispar 1, heated
891 unheated	40	5120	5120	5120	160	640	1280	2560	5120	5120
891 heated	-	5120	5120	5120	160	5120	40	40	5120	5120
2499 unheated	160	5120	5120	5120	320	1280	160	320	40	2560
2499 heated	-	5120	5120	5120	80	320	-	-	2560	2560
3162 unheated	40	5120	5120	5120	2560	5120	320	1280	2560	2560
3162 heated	40	5120	320	5120	5120	5120	80	80	2560	2560

Absorption with *S. dysenteriae* 1 or with A-D 01 removes the inhibition of the agglutination with the unheated homologous and heterologous type organisms to varying extents (tables 2 and 3). Absorption with A-D 01 demonstrates a relationship between these organisms by causing, in most cases, a marked decrease in the

titer. Absorption by a combination of these two antigens causes a greater lowering of titers.

TABLE 2. *Titers of unabsorbed and absorbed antisera prepared against heated strain 891*

Test antigen	Absorbing antigen						
	Unabsorbed	S. dysenteriae 1	Alkalescens-Dispar 01	2499 unheated	2499 heated	3162 unheated	3162 heated
891 unheated	-	5120	1280	-	20	80	-
891 heated	5120	5120	320	-	20	-	-
2499 unheated	5120	1280	640	-	80	-	-
2499 heated	5120	5120	640	-	-	-	-
3162 unheated	160	40	-	-	-	-	-
3162 heated	5120	2560	640	-	-	-	-
S. dysenteriae 1	40	-	-	-	-	-	-
Alkalescens-Dispar 01	5120	5120	-	-	-	-	-

TABLE 3. *Titers of unabsorbed and absorbed antisera prepared against heated strain 3162*

Test antigen		Absorbing antigen					
	Unabsorbed	S. dysenteriae 1	Alkalescens-Dispar 01	891 unheated	891 heated	2499 unheated	2499 heated
891 unheated	40	-	-	-	-	-	-
891 heated	5120	2560	640	-	-	-	-
2499 unheated	320	5120	640	-	-	-	-
2499 heated	5120	1280	640	-	-	-	-
3162 unheated	5120	5120	2560	-	-	-	-
3162 heated	5120	5120	2560	-	-	-	-
S. dysenteriae 1	80	-	160	-	-	-	80
Alkalescens-Dispar 01	2560	5120	-	-	-	-	20

Reciprocal absorptions of the 891 type sera removes the antibody more efficiently than absorption with *S. dysenteriae* 1, or A-D 01 alone or absorption with both. However, strain 891 possesses a minor antigen not possessed by 2499. Nearly all *S. dysenteriae* 1 reacting sites and a large percentage of the A-D 01 sites are removed by reciprocal absorptions with the type organisms. These reactions indicate there are some specific antigenic differences among the three related types.

Agglutination tests with antigens of the Flexner and Boyd groups were negative except for the nonspecific Flexner Y. There were minor cross reactions with A-D 02 and A-D 03.

In some further examinations it was found that strain 891 has antigens related to *Escherichia coli* strains 01, 064, and 069. Strain 891 effectively removes agglutinins from antisera prepared against *Esch. coli* strains 01a and 064 (table 4). Absorptions were not carried out on coli 069 antiserum as the initial agglutination was of very low titer in 891 serum or when 891 was tested in this serum.[7]

TABLE 4. *The effect of reciprocal absorption upon the titer of antisera prepared against strain 891 and* Escherichia coli *strains 01a and 064*

Serum	Absorbing antigen	Test antigen				
		Strain 891	S. dysenteriae 1	Alkalescens-Dispar 01	Esch. coli (US-41)	Esch. coli 064
Strain 891	None	20,480	640	20,480	20,480	2,560
	Esch. coli US-41	1,280	-	-	-	-
	Esch. coli 064	10,240	-	*	*	-
Esch. coli US-41 Ola, 1b	None	20,480	80	20,480	20,480	320
	Strain 891	-	-	-	40	-
Esch. coli 064	None	20,480	-	1,280	1,280	20,480
	Strain 891	-	-	-	-	20,480

*Titers not determined, but show complete agglutination by slide agglutination.

DISCUSSION

It can be expected that in the examination of large numbers of isolates from an epidemic disease a small percentage of the cultures will be aberrant. The three organisms described here represent a group of 10 cultures received in this laboratory during the summer and fall of 1951.

The organisms described in this report do not comply with the description of any currently described member of the genus *Shigella,* although based on preliminary cultural characteristics they appeared to be members of this genus.[8] Upon further investigation, it was found that the organisms possessed antigenic relationship with A-D 01 and *S. dysenteriae* 1, although the biochemical reactions conformed to neither of the antigenic types. Subsequent serologic studies indicated the organisms to be more closely related to A-D 01 in that major antigens were shared. On the other hand, only minor antigenic relationships between the 891 type and *S. dysenteriae* 1 occurred. The antigenic relationship of the 891 type to *S. dysenteriae* 1 is the same as that of A-D 01 to *S. dysenteriae* 1. As would be expected, approximately the same degree of cross agglutination was encountered between the 891 type and *Esch. coli* 01 a,b as with A-D 01.[7]

Ewing[9] has pointed out the necessity for considering the serologic results of the various tests as more important than the biochemical, while not allowing the systematics to become so cumbersome as to be impractical. There have, however, been numerous reports concerned with the antigenic heterogeneity of the genus *Shigella* and related groups. Mackie[10] found *Esch. coli* to cross.react with various members of the Flexner and Sonne groups. Stuart and associates[11] described major antigenic interrelationships between *Esch. coli* and the A-D 01 as well as between paracolon bacilli and A-D 01. A minor relationship was observed between A-D 01 and *S. flexneri* serotypes. Ewing, Hucks, and Taylor[12] reported a cross relation between the *Shigella*-like organism (703) and *Esch. coli* 0112 a,b. Ewing[13] reported a wide range of interrelationships between the various serotypes of *Shigella* and the members of the Alkalescens-Dispar group and the 0 group serotypes of *Esch. coli.*

Wheeler and Stuart,[14] in a study of the mannitol negative group of the genus *Shigella,* found there was some degree of agglutination of A-D 01 in *S. dysenteriae* 1 antiserum. The 14 strains of A-D 01 tested were agglutinated to appreciable titers in 7 of the 8 *S. dysenteriae* 1 antisera and to a low titer (1:40 - 1:80) in the eighth antiserum.

The exact taxonomic position of this group of organisms is not clear. Ewing and Kauffmann[15] have made the suggestion that no new serotypes be added to the Alkalescens-Dispar group of the genus *Escherichia,* but that new serotypes be classed as

anaerogenic, nonmotile coliform organisms. As there is an antigenic fraction which is not removed by absorption by A-D 01, these strains probably can not be grouped among the A-D 01, but should be placed with the anaerogenic, nonmotile coliform organisms. This disposition would also take into account the differences in biochemical activity. However, in view of the close antigenic relationship to A-D 01 it is proposed that this group of organisms be identified as Alkalescens-Dispar 01 var. Koji, this designation being a provisional one.

SUMMARY

A group of organisms, provisional serotype Alkalescens-Dispar 01 var. Koji, related to *Shigella dysenteriae* 1 and A-D 01 serologically, but differing in biochemical reactions, has been described. The taxonomic position of these organisms has been discussed in the light of the current views on the systematics of the genera, *Shigella, Escherichia,* and the Alkalescens-Dispar group.

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REFERENCES

1. Hardy, A. V.; Mason, R. P.; and Martin, G. A.: Dysenteries in armed forces. *Am. J. Trop. Med.* 1: 171-175, Jan. 1952.

2. 406th Medical General Laboratory: *Annual Historical Report, 1951.* pp. 141-146.

3. 406th Medical General Laboratory: *Annual Historical Report, 1952.* pp. 161-172.

4. Wood, A. J.; Baird, E. A.; and Keeping F. E.: Primary division of the genus Shigella based on trimethylamine test. *J. Bact.* 43: 106-107, July 1943.

5. Stuart, C. A., and Rustigian, R.: Further studies on Eijkman reactions of Shigella cultures. *J. Bact.* 46: 105-106, July 1943.

6. Ewing, W. H.: *Simplified Methods for the Serological Identification of Shigella Cultures.* Communicable Disease Center, U. S. Public Health Service, Atlanta, Ga. U. S. Government Printing Office, Washington, D. C., 1950.

7. Davis, B. R.: Personal communication.

8. Breed, R. S.; Murray, E. G. D.; and Hitchens, A. P.: *Bergey's Manual of Determinative Bacteriology.* 6th edition. The Williams & Wilkins Co. Baltimore Md., 1948.

9. Ewing, W. H.: Mannitol negative varieties of Shigella flexneri serotypes. *J. Immunol.* 72: 404-410, May 1954.

10. Mackie, T. T.: Specificity of agglutinin reaction for Shigella dysenteriae; agglutinin absorption relationship between Shigella dysenteriae and Escherichia coli. *J. Bact.* 37: 27-50, Jan. 1939.

11. Stuart, C. A.; Rustigian, R.; Zimmerman, A.; and Corrigan, F. V.: Pathogenicity, antigenic relationships and evolutionary trends of Shigella alkalescens. *J. Immunol.* 47: 425-437, Nov. 1943.

12. Ewing, W. H.; Hucks, M. C.; and Taylor, M. W.: Interrelationship of certain shigella and escherichia cultures. *J. Bact.* 63: 319-325, Mar. 1952.

13. Ewing, W. H.: Serological relationships between Shigella and coliform cultures. *J. Bact.* 66: 333-340, Sept. 1953.

14. Wheeler, K. M., and Struart, C. A.: Mannitol-negative Shigella group. *J. Bact.* 51: 317-325. Mar. 1946.

15. Ewing, W. H., and Kauffmann, F.: New coli O-antigen group. *Pub. Health Rep.* 65: 1341-1343. Oct. 13, 1950.

USE OF CAPILLARY TUBING FOR APPLYING ACID IN VERRUCA THERAPY

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IN the past 20 years the writer has run the gamut of technics in the treatment of verruca plantaris—potential cautery with acids, cryotherapy, curettage under local anesthesia, et cetera. In search of an efficient method of applying strong acid solutions to plantar warts, the writer first pursued a technic at the U. S. Naval Training Center, Bainbridge, Md. which, further developed at this dispensary, has produced excellent results. The method allows for complete control of the acid employed in regard to quantity used, penetration into the tissues, and application to the very marginal limits of the lesion.

I prefer to use trichloracetic or bichloracetic acid (saturated solutions) as the destructive agent. These two acids, although strong enough to devitalize the tissues, do not cause the violent reaction sometimes produced by monochloracetic acid.

The superficial cornified layers of the verruca are first pared off with a scalpel as closely as possible, but with the production of little or no bleeding. Then instead of applying the strong acid solution to the surface of the lesion with the customary pointed wood applicator, a standard small glass coagulation capillary tube (75 mm long; 1.0 mm inside diameter) is employed.

The trichloracetic acid solution will rise inside the tiny tube by capillary action when the tip end of the tube is immersed in the bottle of acid. By observing the level of the liquid in the tube, it is simple to determine how much of the acid is being used. I generally employ an acid column in the coagulation tube of about 3/4 inch in height. A large drop of the trichloracetic acid solution may be noticed hanging at the very tip or flowing down along the side of the glass tube upon its withdrawal from the bottle. This should be wiped off with a quick stroke against a towel, paper, or gauze sponge.

From U. S. Naval Dispensary, Department of the Navy, Washington, D. C.

When a pointed wood applicator is used to apply acid to the skin, some of the acid is absorbed by the wisp of cotton that may be wound about the applicator tip and also by the wood of the applicator itself. Some control in applying the acid to the tissues is thus lost. This absorption of acid cannot occur when using the glass capillary tube as the applicator.

Proceeding with the technic, the tip end of the tube is brought to bear against the surface of the verruca. On contact with the tissue, a small amount of trichloracetic acid will leave the tube and moisten the surface of the lesion. As in the well-known manner of pricking the verruca with a pointed wood applicator, the capillary tube is repeatedly pressed against the tissue. As this procedure is pursued, the acid will continue to emerge slowly and will be seen to settle lower and lower in the tube until it is completely spent. If at any time it is desired to expel any or all of the acid from the tube, the tip end can be touched against a towel or gauze sponge. This maneuver will withdraw liquid from the tube in the manner of a blotter.

The repeated pricking of the lesion with the tip of the tiny glass tube will puncture the surface of the verruca and thus permit deeper penetration of the acid. Due to the rapid action of the trichloracetic acid at the same time, the disintegration of the tissue is readily observed during the application. Furthermore, the use of the capillary tube technic permits application of the acid to the very margins of any verruca, regardless of configuration.

In some cases a pause of a minute or so in the procedure is desirable, to allow destructive action in the tissues to proceed. After this momentary wait, resumption of the pricking process for another minute or half a minute will produce greater amounts of destruction of involved tissue.

Some slight pain that may be experienced during the application will generally cease immediately upon termination of the actual pricking of the tissues. At worst, when considerable acid is applied at one sitting to cause rapid destruction of the growth, a stinging sensation may persist for an hour or so following treatment. This pain factor can be controlled by the operator, because he decides whether to apply only one, or more than one capillary tube "dose" during the course of an application.

Following treatment the verruca is covered with a dry dressing, usually an adhesive bandage, and the patient is advised to keep the lesion dry for the first day or two. The second treatment—curetting the devitalized tissue and reapplying the acid—usually is given 8 to 10 days later. If necessary, further treatment can be given after a similar time interval.

Generally, after a few treatments the patient will state that the verruca is no longer painful. The lesion will appear dehy-

drated and devitalized; the overlying tissue may be dark in color. Usually the acid is not reapplied at this time, and the patient should be scheduled for re-examination in two weeks. If after two weeks some wart tissue still remains, the verruca should be touched up again as the occasion demands.

Only three or four applications of the trichloracetic acid have been required to eradicate most of the verrucae treated. Some cases have been cleared after only two applications and some after only one. In two of approximately thirty cases treated, five applications of the acid were necessary for complete eradication.

The method of application described has been used successfully in the treatment of both single and multiple verrucae. It has been employed not only in cases involving previously untreated verrucae, but also in those cases that have recurred following previous treatment. It appears to be a valuable technic for the treatment of the small satellite type of wart which sometimes appears following surgical removal of a larger verruca. The method has not been attempted in those cases of verruca that have recurred following previous treatment by roentgen radiation.

During the procedure of pricking the verruca, clogging of the tiny capillary tube, caused by a backing up of some of the devitalized tissue, may occur. In such event the tube should be discarded for another or the clogged portion may be broken off by holding the clogged part of the tube between the folds of a 4- by 4-inch gauze sponge. After fracture, the fresh end of the tube should be wiped clean with a stroke of the sponge.

Capillary tubes generally are stocked in two lengths, 75 mm and 150 mm, packed 100 to a container. The cost is very nominal. Tubes 150 mm or longer may be broken to desired lengths. The tip end of a broken length of tube always should be wiped clean before use. Incidentally, the sharp end of a broken length of capillary tubing will more easily puncture the surface of the verruca, permitting better penetration of the acid. Under control of one skilled in the technic, this is a desirable feature.

This technic may have value in other dermatologic procedures. Variations of the technic and other effects will become apparent during its use.

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AN ALKALINE TELLURITE LAURYL SULFATE-SALT PLATE FOR THE ISOLATION OF VIBRIO COMMA

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YOSHIKAZU WATANABE, *M. D., Ph. D.*

MANY media have been described for the isolation of cholera vibrios. Some, while highly efficient, are difficult to prepare, especially under field conditions. Others, like the plate of Aronson and plain alkaline agar, do not require special ingredients or elaborate procedures. Their selectivity is based on their high pH value, usually 9.2 to 9.6. The Aronson medium contains sucrose and starch as fermentable substances, and Andrade's indicator.[1] The brand of starch used in this plate greatly influences the development of the color of the colonies. In addition, many other intestinal organisms will also grow on plain alkaline agar and on the Aronson medium. The most troublesome are Pseudomonas, Alcaligenes, and certain coliforms which, furthermore, may carry partial antigens of cholera vibrios. Proteus and other Enterobacteriaceae frequently spread on the surface of freshly prepared alkaline media. Thus our desire was to find proper inhibitory substances for these basically excellent plating media in order to facilitate the primary isolation of cholera vibrios on them by the less experienced. Moreover, a way was sought to utilize ingredients which are present in most small laboratories equipped with American media and chemicals.

The first inhibitory substance considered was potassium tellurite, used successfully by Gohar and Makkawi[2] in their enrichment fluid during the 1947 cholera outbreak in Egypt. To our knowledge, it has not been tried as yet in solid media. It is readily available in the form of tellurite-blood solution (Difco Laboratories or Baltimore Biological Laboratory) which is used for the preparation of diphtheria plates. This solution consists of defibrinated beef blood with 1 per cent potassium tellurite.

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The second ingredient was sodium lauryl sulfate which is widely used in a broth for the detection of coliforms.[3] Sodium lauryl sulfate was shown, by Bayliss and Halvorson,[4] to have germicidal properties and to clear cultures, including those of vibrios.[5] In the present experiments, it was used in a 0.1 per cent aqueous solution.

Venkatraman and Ramakrishnan[6] advocated the use of sea salt in liquid media exceeding the concentration of sodium chloride employed in most medical bacteriologic media, *i. e.*, 0.5 per cent. An increase of the salt concentration was, therefore, also included in this study.

Finally, bile and bile salts are frequently incorporated into cholera media. In the experience of these writers, vibrio colonies grown on such media are often larger than those grown on plain agar or on Aronson's culture medium. Ox bile (Armour) was used in our comparative studies.

As the base for the media, blood agar base (Difco or B. B. L.) was used. This is available in the majority of laboratories. It incorporates a sufficient amount of a peptone which greatly favors the growth of vibrios.[1] It contains 1 per cent tryptose or thiotone, 0.5 per cent sodium chloride and beef heart infusion. Preliminary experiments showed that autoclaving of the base is not necessary if the inhibitory agents are added immediately and plates are poured without delay.

As test organisms, two freshly isolated strains each of Inaba, Ogawa, and El Tor vibrios were used. *Aerobacter aerogenes, Escherichia coli, Proteus morgani, Pseudomonas aeruginosa,* and *Bacillus subtilis* strains were selected to make up the "test flora" or the "contaminating flora." The strains chosen for this experiment grew vigorously on plain agar. The Proteus and the Aerobacter used throughout this investigation had a tendency to spread over the surface of the plates to a certain extent. This was probably due to the changes in the pH of the media resulting from the utilization of some ingredients by these organisms.

The test strains were kept on agar slants and inoculated into separate broth tubes the day before use. After overnight incubation at 37°C, the cultures were washed off with saline and standardized to contain 100,000 to 120,000 organisms per ml. For the simple growth test, 0.1 ml of each suspension was dropped on the surface of separate plates and spread out using the clock streak. When plates with artificial stools were examined, equal volumes of standardized suspensions of the "contaminating flora" were mixed with the respective Vibrio culture, and the plates inoculated in the same manner as for the simple growth test.

The evaluation of the results was based on (1) inhibition of organisms other than vibrios, especially the blocking of spreaders; (2) colony size; and (3) serologic reactions of the Vibrio colonies.

Colony size was determined by averaging the diameters in millimeters of 10 isolated colonies. Serologic testing was carried out by the plate method, using single-factor sera prepared in this laboratory.

In preliminary experiments, ox bile was excluded. It did not inhibit vibrios but stimulated the growth of *A. aerogenes*, Proteus and Pseudomonas to such an extent that they interfered with the easy isolation of vibrios. While bile and bile salts are known to enhance the multiplication of *Esch. coli*, this was not noticeable with the strains used in these experiments.

Neither tellurite-blood solution nor sodium lauryl sulfate could inhibit completely the growth of the contaminating flora in concentrations to 0.2 per cent tellurite-blood solution and 0.01 per cent sodium lauryl sulfate separately or in combinations. These amounts were the maximums tolerated well by vibrios without a diminishing of the colony size. Using different concentrations of sodium chloride, sucrose as a carbohydrate source, and changing the pH, the results shown in table 1 were obtained.

Table 1 shows that this combination plate is most selective when made up as follows, with sodium chloride concentration of 1.5 per cent, which is the average of the concentrations shown in the eighth and ninth columns under pH 9.6.

Dissolve blood agar base (Difco or B. B. L.) .....40 grams
in distilled water ..... 800 ml
by heating in a boiling water bath. Add
Sodium chloride ..... 15 grams
Shake until dissolved, then add
10 per cent sodium carbonate ..... 50 ml
Check pH. It should be 9.6. Then add
20 per cent sucrose ..... 50 ml
0.1 per cent sodium lauryl sulfate ..... 100 ml
Let cool to about 75°C, then add
Tellurite-blood solution, (Difco or B. B. L.) ..... 2 ml

Keep at 75°C for a few minutes, then cool to about 50°C and pour into petri dishes, about 20 ml per dish.

The plates should be used within three days. They have to be stored in a cool place.

TABLE 1. *Growth of vibrios and "contaminating flora" on media of different composition*

	Concentration, in per cent or value																							
pH	8.4		9.2										9.6										Aronson	Alkaline agar
Tellurite-blood solution	0.2	0.2	0.2	0.2	0.2	0	0	0	0.1	0.2	0.2	0.2	0	0	0	0.1	0.1	0.2	0.2	0.2	0.2	0.2		
Sodium lauryl sulfate	0.01	0.01	0.01	0.01	0.01	0	0.005	0.01	0	0	0.005	0.01	0	0	0.005	0	0.005	0	0.01	0.01	0.01	0.01		
Sodium chloride	0.5	0.5	0.5	0.5	1.0	3	3	3	3	3	3	5	0.5	0.5	0.5	0.5	0.5	0.5	0.5	1	2	3		
Sucrose	0	1	0	1	0	0	0	0	0	0	0	0	0	1	1	1	1	1	1	1	1	1		
Inaba	*2.5	2	2.5	2.5	2	3	3	2.5	3	2.5	1.5	0	2	3	2.5	2.5	2.5	2	2.5	2.5	2.5	1.5	3	3
Ogawa	2.5	2	2.5	2.5	2.5	2.5	2.5	2.5	2.5	2	3	0	2	3	2	2	2	2	2	2.5	2.5	1	3	3
El Tor	2.5	3	2.5	3	3	2	2	2	2.5	2	2	0	2.5	3	2	2	2	2	2	2.5	2	1.5	3	3
Esch. coli	0	0	0	0	0	1.5	1.5	1.5	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	3
A. aerogenes	2s	2s	2s	2s	1.5	2S	2s	2	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	3S
Proteus	1.5s	1.5s	1.5s	1.5s	1	2S	2s	2.5	1.5	1.5	0	0	2.5	2.5	1.5	2	0	2	0	0	0	0	2	3S
B. subtilis	1	2	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2
Pseudomonas	1	2	1	2	0	1.5	1	1	1	1	0	0	1	1	0	0	0	0	0	0	0	0	1	3
Vibrios with contaminating flora													S	S	S	sd	sd	sd	2.5 e	2.5 e	2.5 e	2.5 e	sd	S

* – Colony size in mm after 24 hours' incubation adjusted to nearest 0.5 or whole number
s – Spreading colonies
S – Strongly spreading growth
d – Vibrios difficult to isolate
e – Vibrios easy to isolate

One-hundred fifty Vibrio strains were tested on this medium. All of the 31 Inaba cultures, 44 of the 46 Ogawa strains, 25 of the 26 El Tor types, 4 of the 5 Hikojima organisms, and 9 of the 12 "nonagglutinable" vibrios grew well on the plate. The strains which failed to grow on the new medium were old stock cultures, while recent isolates multiplied vigorously.

Of the stock organsims which were not cultivable on the new plate, 1 Ogawa, 1 Hikojima, and 2 "nonagglutinable" vibrios, however, formed colonies on Aronson's plate.

When selected colonies grown on the new medium were typed with specific sera, the slide agglutination test gave rapid positive reactions in all cases.

All *Vibrio comma* showed translucent, grayish colonies on the new medium in 20 to 24 hours, which turned blacker after longer incubation. Whitish gray colonies also were produced by *A. aerogenes* which, however, needed 40 to 48 hours to develop the gray discoloration. Aerobacter growth was always opaque, in contrast to the translucent Vibrio colonies. It is believed that this reduction of potassium tellurite is helpful in the recognition of vibrios. Experiments are being set up to check other intestinal organisms, especially Enterobacteriaceae, for their ability to give the same reaction.

Thirty stools, received for routine examination from cases of diarrhea not involving vibrios, were streaked to the new plate. They showed no growth of any organism in 24 hours.

Finally, the new plate was compared with Aronson's and with plain alkaline agar, plating to it either vibrios alone or a mixture composed of an equal number of a Vibrio, an *Esch. coli,* an *A. aerogenes,* a *Pr. morgani,* a *Ps. aeruginosa,* and a *B. subtilis* strain described in previous experiments. Measured amounts of this mixture, to contain 3, 30, and 300 vibrios per inoculum (0.1 ml), were streaked to 10 plates each. The results are shown in table 2, as determined after 24 and 48 hours' incubation.

The new plate proved itself very selective after 24 hours' incubation. The contaminating flora did not grow in 24 hours. It became evident only after 48 hours, while Vibrio colonies still could be selected from the surface of the media with ease. Vibrios formed smaller colonies on the new plate but the contaminating flora was suppressed to a great extent (table 2).

Admittedly, the plates were subjected to too severe tests by using a contaminating mixture composed of several nonfastidious organisms, and checking against small numbers of vibrios. It is believed, however, that such testing will produce results which are less likely to crumble under the impact of field evaluation.

TABLE 2. *Comparison of colony size of vibrios on three media*

Strain tested	Incubated hours	Expected number of organisms in inoculum									
		300			30			3			
		Number of vibrio colonies observed/average diameter of colonies in 3 mm									
		Aronson	Alk. agar	New plate	Aronson	Alk. agar	New plate	Aronson	Alk. agar	New plate
Inaba	24	400/2.5*	350/2.5	360/2	33/3**	30/3.5	34/2	4/4	4/4	4/2
Ogawa	24	300/2.5	320/3	290/1.5	29/3	29/3	31/2	3/3	3/3	3/2
El Tor	24	280/3	280/3	300/2	23/3	24/3.5	28/2	2.5/3	3.5/3.5	3/2
Vibrios plus contaminating flora:										
Vibrios	24	Sn	Sn	330/1.5	Sn	Sn	35/2	S1/2	Sn	3/2
	48	Sn	Sn	330/2	Sn	Sn	35/2.5	Sn	Sn	3/2.5
Contaminants	48	S	S	72/<0.5	S	S	3/<0.5	S	S	0/0

Alk. - Alkaline
* - Adjusted to nearest 10
** - Adjusted to nearest whole number
n - Vibrios not isolated
S - Strongly spreading growth

The new plate is not suggested as a sole medium for the isolation of vibrios. It appears to be feasible for use together with plain alkaline agar and, if possible, with Aronson's medium. It permits the growth of El Tor vibrios, in which aspect it differs from the excellent medium of Wilson and Reilly.[7] Although it is not believed to be superior to the Panja and Ghosh plate,[8] it is much easier to prepare. While most authorities agree that Panja's[9] method of filtration of the stools through a porcelain candle into a boric acid medium has not been surpassed by any other method of cholera culture,[10] the difficulties involved in filtering each specimen for several hours compel us to use plating media inoculated directly as well as from enrichment or preserving broths. Improvement and further development of the cholera plates are needed yet.

SUMMARY

A medium consisting of commercial blood agar base with 0.01 per cent sodium lauryl sulfate, 0.1 per cent tellurite-blood solution (equivalent to 1:100,000 potassium tellurite), a final sodium chloride concentration of 2 per cent, and 1 per cent sucrose at a pH 9.6, was described. This plate is feasible for the growth of *Vibrio comma* but inhibitory for other organisms commonly found in the intestinal tract. The grayish discoloration of the Vibrio colonies developing on this medium facilitate their recognition.

ACKNOWLEDGMENTS: The authors are most indebted to Professor G. Panja from the School of Tropical Medicine of Calcutta; Professor K. C. Basu Mallik and Dr. A. Mondal of the Nilratan Sircar Medical College of Calcutta; Dr. E. W. Soman, Director of the Haffkine Institute of Bombay (Ret.); and Mr. S. T. W. Tanamal, Bacteriologist of the Public Health Laboratory in Makassar for the strains used in these experiments.

REFERENCES

1. Felsenfeld, O.; Soman, D. W.; Waters, T.; and Ishihara, S. J.: Studies on recently isolated cholera vibrios; re-evaluation of culture methods. *J. Bact.* 62: 175-180, Aug. 1951.

2. Gohar, M. A., and Makkawi, M.: Cholera in Egypt; laboratory diagnosis and protective inoculation. *J. Trop. Med.* 51: 95-99, May 1948.

3. Mallmann, W. L., and Darby, C. W.: Use of lauryl sulfate tryptose broth for the detection of coliform organisms. *Am. J. Pub. Health* 31: 127-134, Feb. 1941.

4. Bayliss, M., and Halvorson, H. O.: Germicidal and detoxifying properties of soaps. (Report of paper read) *J. Bact.* 29: 9-10, Jan. 1935.

5. Bayliss, M.: Visible action of sodium lauryl sulphate on microorganisms. *J. Lab. & Clin. Med.* 22: 700-704, Apr. 1937.

6. Venkatraman, K. V., and Ramakrishnan, C. S.: Preserving medium for transmission of specimens for isolation of Vibrio cholerae. *Indian J. M. Research* 29: 681-684, Oct. 1941.

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7. Wilson, W. J., and Reilly, L. V.: Bismuth sulphite media for isolation of V. cholerae. *J. Hyg.* 40: 532-537, Sept. 1940.

8. Panja, G., and Ghosh, S. K.: Modified medium for isolation of dysentery, enteric and cholera organisms. *Indian M. Gaz.* 78: 43-46, Jan. 1943.

9. Panja, G.: New method of isolation of vibrios from cholera stools. *Indian J. M. Research* 30: 391-396, July 1942.

10. Pollitzer, R.: Cholera studies; bacteriology. *Bull. World Health Organ.* 12: 777-875, 1955.

MODERN MEDICINE MAN ON TELEVISION

"Along with many of the excellent reforms in medicine that took place soon after the turn of the century was a definite attempt to educate manufacturers, editors and the public regarding the dangers of advertising and selling medicines direct to the public. . . . Not all editors or pharmaceutical manufacturers accepted these reforms, but the influence of those who disregarded them was greatly lessened and they were increasingly looked upon with disfavor. Unfortunately, those who continued direct dealings with the public soon began using the medium of radio for advertising and as the years went by, radio became a raucous voice plugging sedatives, headache powders, vitamins and numerous other medicinals until the ears of the public rang with the constant din of its chatter. After radio came television, which has brought the ancient medicine show to life. Just as the credulous grandfathers of the present generation found the Indian medicine man and old Doc Jones and his cure for rheumatism convincing, so do their grandchildren find "tired blood," "relieves headache twice as fast," "like a doctor's prescription" and other ridiculous statements, claims, half-truths and the associated entertainment convincing to an incredible degree. Unfortunately, the same factors are operating in the modern version as with the old Doc Jones show, but where the previous generation shelled out two bits to a dollar for a bottle, the grandchildren frequently shell out five dollars and not infrequently come back for more and continue to do so for months."

—EDITORIAL
in *The New England Journal of Medicine*
p. 248, Jan. 30, 1958

A SELF-CLEANING DENTAL MIRROR

WALTER M. ORMES, Jr., *Major, DC, USA*

A distinguished oral surgeon stated during a series of lectures, "In any oral surgical procedure the operator must see well what he does to do well what he sees." This statement should never be forgotten, not only in oral surgery, but in all fields of dentistry.

With the advent of the water-spray attachment for the dental handpiece, and then the high-speed handpiece, the ordinary dental mirror became practically useless. An operator had the choice of working without the water spray and thus risking thermal damage to the pulp, or using the water spray and viewing the field of operation through a mirror showing a distorted image due to water and debris.

Operating without the water spray was the solution with a slow-speed handpiece, but with the high-speed handpiece the cooling effect of the spray is a necessity to protect the pulp. It became apparent that the solution to the problem must lie in a mirror that would remain clear, while operating with the water spray attachment.

The idea occurred that if a stream of air could be made to strike the surface of a mirror, the force of the air blast would blow away the water and debris and result in a clear image. Using a piece of plastic tubing 3 mm in diameter, a metal tip to fit the nozzle of the spray-bottle air syringe, and another metal tip to rest against the mirror and held in place with copper bands, a fairly satisfactory air-dried mirror was developed. This mirror, although better than an ordinary mirror, left much to be desired. The metal tip adjacent to the mirror, being round, would not properly direct the flow of air; the copper bands had a tendency to slip, causing the tip and tubing to twist out of position; and the instrument could not be sterilized without taking the bands off, removing the plastic tubing, and then reassembling after sterilizing.

Eventually, the mirror illustrated in figure 1 was developed with the help of the Third Army Ordnance Section. The disassembled instrument is shown in figure 2. The easily removable mirror section is fabricated from cast aluminum, with a flat tip

From Dental Detachment, U. S. Army Garrison, Fort McPherson, Ga.

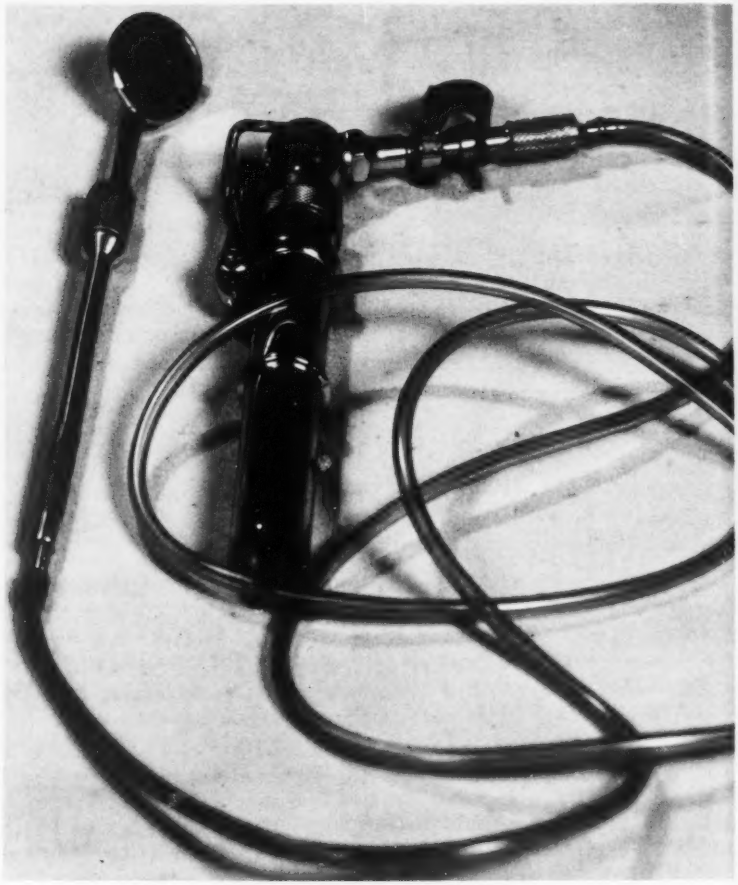

Figure 1. The self-cleaning dental mirror assembled.

adjacent to the mirror proper so that the stream of air will sweep the entire surface of the mirror. The handle of the mirror is a hollow tube for passage of the air blast, thus eliminating the copper bands used in its prototype. The plastic tubing slips off the end of the mirror handle easily, permitting sterilization of mirror and handle as a unit. The tube with tip for attachment to the air syringe, shown in figure 2, and the clamp, shown in figure 3, complete the instrument. This clamp is used to lock the air syringe in the "on" position.

This mirror has been used clinically with a high-speed handpiece with built-in water-spray attachment, and has been found to be satisfactory in both crown-and-bridge and operative pro-

cedures. The air blast from the syringe keeps the surface of the mirror clean. It requires only occasional wiping regardless of whether diamond stones, disks, or burs are used in the handpiece. The noise of the air rushing over the surface of the mirror

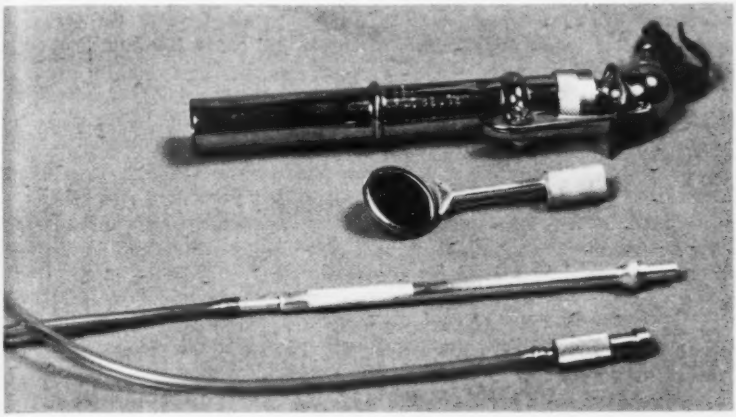

Figure 2. The instrument disassembled.

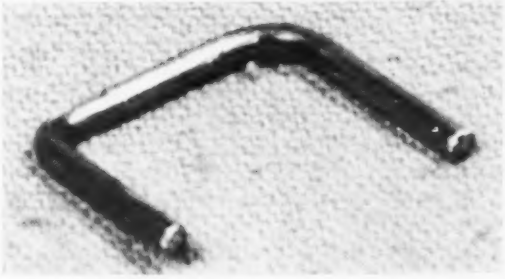

Figure 3. Clamp used to lock air syringe in the "on" position.

is not objectionable to the patient. By tilting the mirror toward the tooth being operated on, the field is dried for better observation in a matter of seconds. The assistant, by not having to operate the air syringe, is freed for other duties.

The mirror handle and tip can be made in any ordnance shop, and the plastic tubing and air-syringe tip are available through Medical Supply. The air-syringe clamp can be made from a nail or piece of heavy wire.

SUMMARY

With the use of water-spray handpiece attachments and high-speed dental handpieces, the conventional dental mirror is practically useless. In order to obtain proper vision, a mirror that is self cleaning must be used. The mirror described above is such an instrument. It is not difficult to manufacture, is satisfactory in clinical use, and has no maintenance or sterilizing problem.

WHY TELL THE PATIENT?

"Standard textbooks of medicine supply excellent articles on recognized diseases, but with rare exceptions the discussions fail to include one feature of paramount importance. No mention is made of the need for explaining to the patient the nature of his problem, the mechanism and significance of his symptoms, and the course the illness may be expected to follow. This omission is unfortunate because, with present-day concentration on pathologic physiology in our medical schools, the subject also receives little or no attention in clinical teaching. As a consequence it may be several years after graduation before a young internist appreciates all that can be accomplished by sufficiently detailed instruction of those under his care. There are two principal reasons why an explanation along the lines indicated is indispensable. In the first place, it is, in many illnesses, a therapeutic tool of basic importance. Secondly, it is one of the best available ways to show personal concern for the patient's welfare and therefore a most effective method for maintaining public support of our free enterprise system of medicine."

—A. CARLTON ERNSTENE, M. D.
in *Journal of the American Medical Association*
p. 1110, Nov. 2, 1957

CHELATING SUBSTANCES IN MEDICINE

ROBERT B. ABINGTON, *Captain, MC, USA*

CHELATING substances have been used in industrial chemistry for many years, either as a means of inactivating a metallic ion or to assist in increasing the solubility of relatively insoluble metallic compounds. The term "chelation," however, was practically unknown to medicine until the end of World War II, when the medical implications of British antilewisite (BAL) were made known. The therapeutic uses of BAL have been limited to the treatment of poisoning or untoward reactions from certain heavy metals; namely, arsenicals, mercurials, and gold salts. Recent developments with newer chelating substances promise to be beneficial in the study and treatment of some of the more common maladies encountered in medicine.

The word "chelate" comes from the Greek word, chēlē, which means claw. This is extremely descriptive of chelating agents in that they possess the unique ability to combine with or "entrap" a metallic ion and inactivate it. In simple terms, the metallic ion reacts with the two ends of a short chained substance, thus creating a ring structure with the metallic ion tightly bound within the ring. In reality the structural mechanism is more complex, but the metallic ion always is bound within the molecular structure of the chelate from at least two sides. With the surrounding and binding down of the metallic ion, it is inactivated and isolated from further chemical reactions. This stable, readily soluble, metal complex then is easily removed from the body by normal renal function.

The theoretical implications of chelates and their effect on body metabolism are practically boundless. Any enzyme reaction utilizing a trace metal could be potentiated or inhibited by appropriate chelates, depending on the degree of attraction for the specific metal that is necessary for the enzyme reaction.

Many of the drugs used in medicine are chelating agents. The list is too long to tabulate, but includes many of the antibiotics and the present-day antituberculosis drugs. The role that chelation plays in influencing the effectiveness of these drugs is poorly understood. A practical point concerning antibiotics is

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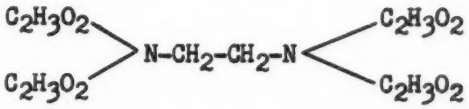
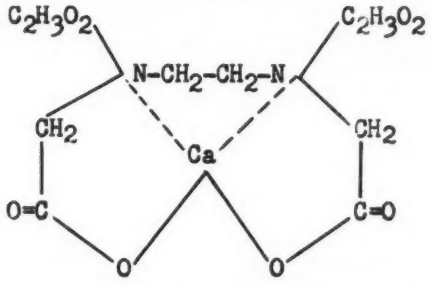
the chelation of aluminum and magnesium by the tetracyclines. The metal-salt combination prevents absorption of the antibiotic from the gastrointestinal tract. The use of antacids containing aluminum or magnesium along with the tetracyclines therefore is considered unwise.

EDTA

Ethylenediaminetetracetic acid (EDTA), the most potent chelating agent available today, was discovered in Germany by Pfeiffer and Schwartzenbach. This relatively simple compound contains four acetic acid groups attached to ethylenediamine. The structural formula is as follows:



This substance has assumed clinical importance for many reasons. It is a potent chelator that is not metabolized and therefore may be used to remove calcium and certain toxic metals from the body. It is relatively nontoxic and inexpensive. In the presence of heavy-metal or alkaline-earth ions, EDTA reacts to form highly stable, soluble, metal-chelate compounds. The structure of the calcium chelate may be depicted as follows:



As with other chelates, EDTA has a variable affinity for different metals, depending on the dissociation or stability constant and the relative concentration of the exposed ions. In general, metals such as copper, nickel, and lead are bound more tightly than those of the alkaline-earth group, including calcium, magnesium, and barium. These alkaline earths in turn are bound to a much greater degree than the alkali metals, sodium, potassium, and lithium. Therefore, a sodium-EDTA complex would be replaced in the body by calcium complex. Likewise, a lead-EDTA complex would be excreted unchanged, in man.

For several years after its release on the American market, EDTA was believed to be a nontoxic drug with a wide margin of safety if used in the recommended dosage, but recent observations prompted a reappraisal of this drug and it now is established that EDTA may result in a toxic nephrosis. The hydropic degeneration of the proximal tubules is thought to be a reversible process if the drug is discontinued early enough.[1] Toxicity is related more to the duration of treatment than to the amount of drug used. The currently recommended dosage is 50 mg per kg for five days, followed by a rest period of at least two days. Daily urinalyses should be carried out if EDTA is used intravenously. The presence of acute or chronic renal disease probably should cause some reluctance in using this drug.

CLINICAL INVESTIGATIONS WITH EDTA

Lead Poisoning. Experimental trial of EDTA in patients with inorganic or organic lead poisoning has passed the investigational stage. It now has been accepted by most authorities to be the drug of choice. Calcium Disodium Versenate (brand of edathamil calcium-disodium) is used to prevent the chelate from lowering the serum calcium. The alkaline metals (Na) and earths (Ca) are easily displaced by the heavy metal (Pb) within the body and excreted as the lead complex of EDTA.

Hypercalcemia. Calcium is present in man in such large concentrations and has such a strong affinity for EDTA that hypocalcemia can be produced easily by injecting the chelate. EDTA therefore has been used in an effort to remove metastatic calcifications, calcium deposits around joints, calcified corneal opacities, calcified vascular plaques, renal calcifications, and other abnormal calcific deposits. Although reports are conflicting, it appears that calcium can be removed successfully[2] if this chelate is used properly over a prolonged period of time. It must be emphasized that the treatment results in only a transient benefit unless the underlying condition is corrected. Judicious use of EDTA seems particularly helpful in alleviating symptoms of hypercalcemic toxicity. Loss of symptoms and return of renal function were noted for several months in patients with hypercalcemia, advanced carcinomatosis, and renal failure.

Coronary Atherosclerosis. The problems of existing coronary atherosclerosis and of how to increase needed blood supply to endangered tissues have been approached with EDTA. It is known that calcium is integrated with the cholesterol in an atheroma, and thereby becomes an active participant in a living pathologic structure. By chelation of the calcium, it is believed that the organic atheromatous matrix might undergo disintegration. A recent study[3] of 20 patients with clinical evidence of advanced coronary atherosclerosis and progressive angina pectoris indicates that multiple infusions of EDTA may reverse this process

in part. It appears that by repeated chelation of calcium within the body the symptoms and signs of vascular insufficiency caused by atheromatous plaques may be relieved for a significant period of time. Although cardiac stress tests were not used, serial electrocardiograms revealed reversion of the ischemic patterns to normal. This correlated with the clinical improvement of all patients. Of the 20 reported patients with angina pectoris, 19 survivors obtained unusual symptomatic relief that could not be attributed to any other source.

The only apparent hazard associated with this study was the possibility of a calcium embolus occurring. Rapid disintegration of an atheromatous plaque may cause fragmentation of the friable calcium-cholesterol matrix and dislodgement into the blood stream. One reported autopsy seemed to confirm this.

The relationship and effect of chelating agents on atherosclerosis need considerable study and evaluation before any conclusions can be drawn.

Treatment of Digitalis Toxicity. Potassium *depletion* or calcium *excess* enhances the toxic effects of digitalis. Likewise, calcium *depletion* or potassium *excess* have been found to prevent or abolish digitalis in induced cardiac arrhythmias. It is reported that injection of a small quantity of EDTA chelates some of the available calcium ions and quickly terminates arrhythmias resulting from digitalis intoxication.[4] It is conceivable that EDTA or a similar chelate could replace potassium when a rapid intravenous medication is needed for cardiac arrhythmias secondary to digitalis toxicity.

Radioactive Decontamination. The majority of fission products of a nuclear explosion have a short half-life and therefore pose no treatment problem, but those with a long half-life present a chronic internal radiation hazard. Chelating substances have been employed to remove these radioactive metals from the body. EDTA and zirconium citrate are presently being tested by research laboratories.[5] The effectiveness of this treatment depends to a great extent on the rapidity of administration of the chelate after exposure. Recent studies indicate that the most effective treatment for exposure to multiple fission products of a nuclear explosion consists of pretreatment with EDTA, followed by zirconium citrate soon after exposure.

INVESTIGATIONS WITH OTHER CHELATES

Radiation of Tumors With Chelates. Uranium^{235} is said to be an excellent substance for the treatment of tumors. Neutron capture by U^{235} releases enormous amounts of energy over very short ranges in the form of fission particles. If fission were to take place in the tumor, the tumor might be completely destroyed.

In the past, clinical trials with this substance have not been possible because administration of this metal in the free state has resulted in serious kidney and liver damage.

Porphyrins are one of the many excellent chelates present in nature. It has been known that porphyrins concentrate heavily in tumors, and in embryonic and inflammatory tissues. Chelation of U^{235} with porphyrin would seem to be an ideal way to transport this substance to inaccessible tumors. U^{235} would then be activated by the neutrons from an atomic reactor. Early experimental studies indicate that the uranium-porphyrin complex does not have the toxic properties of free U^{235}.[6] If this work is confirmed, it is possible that chelates will be invaluable in the treatment of cancer.

Chelation of Ferrous Sulfate. By combining ferrous sulfate with a chelating agent, the almost universal intolerance to Fe has been overcome. Recently a new hematinic, an alpha aminoacetic ferrous sulfate complex, was studied.[7] By chelating iron within the molecular structure of the amino acid, it was found that the absorption of iron was increased markedly[8] and that gastric irritability from the ferric compounds formed in the stomach was prevented.

SUMMARY

Chelating substances react with a metallic ion by encompassing the metal within the molecular structure and inactivating it. The affinity of a chelate for various metals depends on their stability constants and relative concentrations. Ethylenediaminetetracetic acid (EDTA) is the most potent chelate presently employed, and its use is established as a good method for removal of certain heavy metals from the body. Recent clinical investigations of EDTA suggest possible benefit in using it in several different diseases, and the value of chelates as carriers of radioactive metals within the body is presently being studied.

REFERENCES

1. Foreman, H.; Finnegan, C.; and Lushbaugh, C. C.: Nephrotoxic hazard from uncontrolled edathamil calcium-disodium therapy. *J. A. M. A.* 160: 1042-1046, Mar. 24, 1956.

2. Spencer, H.; Greenberg, J.; Berger, E.; Perrone, M.; and Laszlo, D.: Studies on effect of ethylenediaminetetraacetic acid in hypercalcemia. *J. Lab. & Clin. Med.* 47: 29-41, Jan. 1956.

3. Clarke, N. E.; Clarke, C. N.; and Mosher, R. E.: Treatment of angina pectoris with disodium ethylene diamine tetraacetic acid. *Am. J. M. Sc.* 232:-654-666, Dec. 1956.

4. Gubner, R. S., and Kallman, H.: Treatment of digitalis toxicity by chelation of serum calcium. *Am. J. M. Sc.* 234: 136-144, Aug. 1957.

5. Cohn, S. H.; Gong, J. K.; and Milne, W. L.: Experimental treatment of poisoning from fission products. *A. M. A. Arch. Indust. Health* 14: 533-538, Dec. 1956.

6. Bases, R. E.: Uranyl protoporphyrin: new uranium complex. *Science* 126: 164-165, July 26, 1957.

7. Pomeranze, J., and Gadek, R. J.: Clinical appraisal of drugs; with special reference to new chelate hematinic. *New England J. Med.* 257: 73-75, July 11, 1957.

8. Bessman, S. P., and Doorenbos, N. J.: Chelation. (Editorial) *Ann. Int. Med.* 47: 1036-1041, Nov. 1957.

CASE REPORTS

Unilateral Renal Vein Thrombosis

ERNST R. MOELLER, *Captain, MC, USN*

A CASE of unilateral renal vein thrombosis that was successfully treated is presented. While reviewing the literature, it was noted that there were very few case reports in which therapy was instituted and in which the patient survived. In the vast majority of cases the condition was found at autopsy. It therefore was deemed pertinent to present this case, not for the purpose of advocating new theories or treatment but rather to call the condition again to the attention of both pediatricians and urologists. Expedition in instituting therapy after the condition has been considered or verified is the prime factor in decreasing the mortality rate.

CASE REPORT

A Negro male infant, age 6 weeks, was seen in the outpatient department with the history of having had fever for the previous 18 hours, and 2 generalized convulsions during this period. The previous evening, at the onset of illness, he had had a generalized convulsion, and subsequently his mother noticed that he was feverish and listless. He slept fitfully during the night and in the morning had another generalized convulsion, following which his mother brought him into the outpatient clinic.

The mother stated that during the past 48 hours she had noted some blood-tinged urine and some "pink" staining of the diaper. No other symptoms were noted.

Past History

At birth the infant had been premature, weighing 3 lb 13 oz, normal, and in excellent condition. He had been hospitalized for one month and discharged weighing 5 lb. Physical examination at discharge revealed no abnormalities. Progress at home was normal until the onset of the present illness.

Family History

The mother and father were living and well, and there was no history of disease. One sibling, age 3 years, had had convulsions with high fever.

From U. S. Naval Hospital, San Diego, Calif.

Figure 1. Gross appearance of infarcted right kidney.

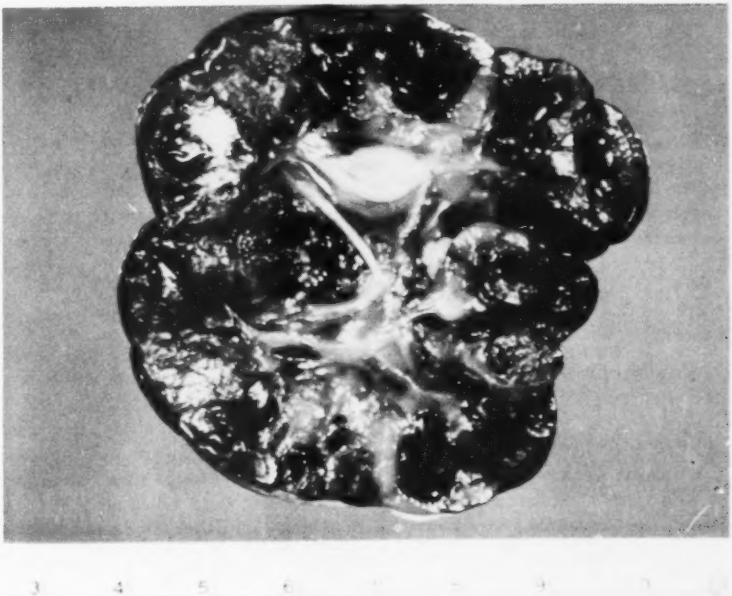


Figure 2. Cut surface of right kidney, showing normal-appearing kidney interspersed between large zones of red-brown tissue.

Physical Examination

On admission to the hospital the infant weighed 5 lb 14 oz. He was mildly dehydrated and acutely ill. His temperature was 101.6°F. The physical examination was negative for unusual findings other than the abdomen, which was moderately distended. In the right flank, extending posteriorly, there was an ill-defined mass about the size of a hen's egg, extending from the costal margin downward and deep into the flank. There was also a mild paraphimosis.

Laboratory Studies

The initial laboratory studies showed the following: red blood cell count, 3,300,000 per μl; hemoglobin, 8.5 grams per 100 ml; and white blood cell count, 15,900 per μl, with 38 per cent neutrophils and 62 per cent lymphocytes. Urinalysis revealed 4-plus albumin, and microscopic examination showed 6 to 8 white blood cells, 4 to 5 finely granular casts, and numerous red blood cells. Radiographic examination of the chest, abdomen, and long bones was negative, as were a spinal tap and a barium enema.

An intravenous pyelogram revealed an apparently normal left kidney and ureter. The picture of the right kidney was obscured, but an area thought to be a single calyx was seen.

Preoperative supportive therapy was instituted, including a blood transfusion, and on the following day exploration was performed. The left kidney and ureter were normal. The right kidney was enlarged, ecchymotic, and boggy. A right nephrectomy was performed.

Pathologic Findings

The specimen consisted of a kidney weighing 33 grams and measuring 5 by 2.5 by 2.5 cm. The capsule stripped with ease. The fetal lobulations were present and the surface was varicolored. There were some areas of normal-appearing kidney on the surface. Between these there were raised, dark red-brown, well-demarcated zones (fig. 1). On cut section the parenchyma had small areas of normal-appearing kidney, interspersed between large zones of red-brown tissue. The line of demarcation between these areas was sharp (fig. 2). Several pyramids were dark red-brown and were well demarcated from the overlying cortex.

Microscopic slides showed large areas of infarction throughout the kidney. Some of these involved only the medulla, while others involved the cortex also (fig. 3). A thin zone of normal cortex remained next to the capsule in those areas where infarction involved the cortex. Some of the infarcted areas showed necrosis with "shadows" of the former structures present, while others were composed of fresh areas of hemorrhagic extravasation with acellular necrosis. There was polymorphonuclear infiltration around the infarcts showing necrosis, but no reaction was noted around the hemorrhagic portions. A thrombus showing evidence of organization was present in the renal vein. There also were multiple thrombi in the smaller veins in normal tissue.

Postoperative Course

After a stormy postoperative 72 hours, the infant tolerated fluids by mouth. The urine was clear on the 8th postoperative day. The blood urea nitrogen and creatinine were within normal limits when he was discharged from the hospital on the 16th day. Several follow-up examinations revealed the infant to be progressing in a normal manner.

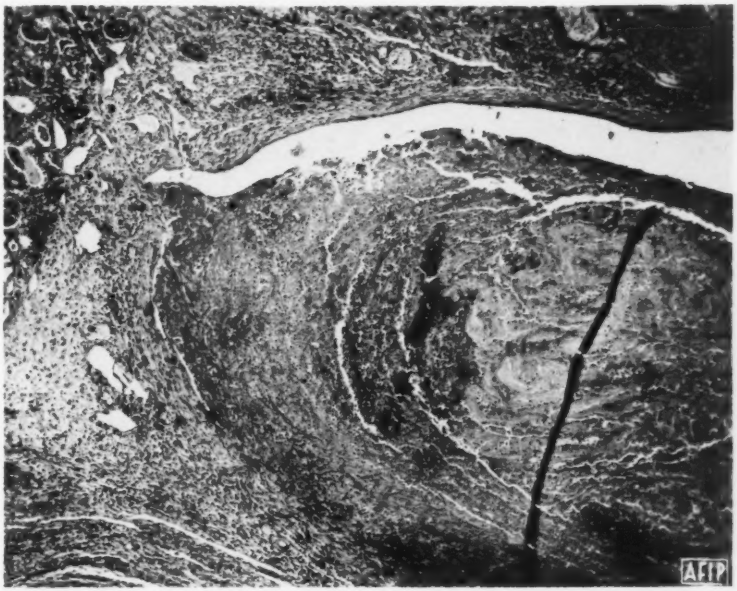


Figure 3. Photomicrograph showing small and large thrombi. (× 48) (Courtesy, Armed Forces Institute of Pathology)

DISCUSSION

The paucity of reported cases of renal vein thrombosis with recovery is evident in reviewing the literature. However, the importance of awareness of this condition is heightened by reports of survival after nephrectomy in at least 12 cases.[1]

The modern doctor usually is not so aware of the entity of acute thrombosis of the renal vein with renal infarction as was his predecessor of the nineteenth century, who regarded thrombosis of the renal vein as a common occurrence in young infants.[2] It is not a common disorder in the pediatric age group, although it is not so rare as a casual survey might lead one to believe. Undoubtedly, many cases pass unrecognized under the inadequate diagnosis of acute septicemia or acute pyelitis. At Babies Hospital (unit of Presbyterian Hospital) in New York City, 43 cases were found in 6,200 autopsies; 32 of these cases were in

the first year of life.[3] McClelland and Hughes[4] encountered three cases of renal vein thrombosis with kidney infarction in 328 consecutive autopsies of infants under one year. In a recent review of the literature, approximately 268 cases were recorded.[5]

The syndrome may occur at any age, but is uncommon beyond the age of 6 months. It has been found equally in both sexes.[3,5] Unilateral involvement occurred in 50 per cent of the cases and there was no apparent predilection to either side. The prognosis was grave in those cases in which both renal veins were thrombosed; however, Fallon[6] reported a recovery in one case. The mortality has been estimated at 95 per cent in unilateral cases without surgery, and 25 per cent with surgery.[7] Death usually occurs 48 to 72 hours after the appearance of the abdominal mass.

ETIOLOGY AND PATHOGENESIS

An initial illness generally is conceded to be a predisposing factor in an overwhelming majority of the cases. In most instances, diarrhea with associated dehydration, acidosis, and collapse preceded or was concomitant with renal infarction. Barenberg and associates[2] reported 5 cases occurring in a series of 25 infants who died in an epidemic of diarrhea of the newborn. Another group[4] found 3 cases in 31 infants who died following severe diarrhea. Although ileocolitis is the principal predisposing factor, other infections that similarly alter the patient's electrolyte balance and hydration could cause renal thrombosis. Measles, scarlet fever, diphtheria, cutaneous infections, and omphalitis were accused in several instances.

Many classifications of this pathologic entity have been advocated.[5,6,8-11] The author believes that a more easily understood classification may be obtained by combining the ideas of Sandblom[8] and Morison:[9]

Class I. Primary thrombosis of the renal veins that occurs in apparently healthy young infants.

Class II. Secondary thrombosis of the renal veins that has been preceded by an acute illness, such as diarrhea. This is the most common variety.

Class III. Thrombosis of the renal veins associated with renal inflammation.

The factors initiating the thrombosis are not known and at the present time we can only theorize as to the possibilities. Stasis of the blood stream undoubtedly is an important factor in intravascular thrombosis. It is well known that the renal tubules of the newborn have relatively little ability to remove water from the glomerular filtrate. This fluid, lost by the glomeruli, is not regained from the collecting tubules. At the same time, the sick infant with vomiting and diarrhea rapidly becomes dehydrated with profound changes in the blood volume as well

as in the blood elements. This results in increased viscosity (hemoconcentration). The uniformly low arterial and capillary pressure and presumably low venous pressure in the neonate, in addition to the nature of the renal circulation with its double capillary network, results in considerable slowing of the stream. Therefore, the normal physiology of the young infant's glomerular system combined with the hemoconcentration, alteration of blood constituents, dehydration, and circulatory stagnation, may well favor intravascular clotting in the renal venules.

Apparently there is no defect in the clotting mechanism in these cases. No evidence has been presented that these infants are more susceptible to hemorrhagic phenomena than the normal newborn. Hepler[10] and Abeshouse[5] believed that the thrombus is formed by direct injury to the intima of the renal vessels, *i. e.*, bacterial, mechanical, chemical, or toxic. These theories could explain the formation of the renal vein thrombosis in the sick infant, but it is difficult to apply them to the apparently healthy infant who suddenly becomes ill with this entity. Perhaps these infants have some intrinsic congenital anomaly of the renal system as the etiologic factor. This was suggested by Warren, Birdsong, and Kelley.[12]

PATHOLOGY

There is little agreement as to whether the smaller veins or the larger branches of the renal vein are first involved. Zuelzer[11] believed that in infancy the thrombosis is primary in the small radicles and may extend secondarily into the main branches of the renal vein or occur simultaneously in branches of small and large caliber. He found no evidence to indicate that retrograde extension of the thrombosis from the main stem of the renal vein played a part in the development of the process. Morison[9] expressed a similar viewpoint. Barenberg[2] reported two cases that had thrombi only in the main veins. The general consensus of opinion, however, is that both small and large veins are involved early and that the main vein is involved with the appearance of symptoms.

Infarction may or may not be present with a thrombosis, depending on the degree and rate of progress of the thrombosis. When massive infarction occurs, the kidney is soft, boggy, and deep purple or chocolate in color. Subcapsular hemorrhage with ecchymosis or hematoma usually is found. In localized thrombosis, the segment involved is demarcated sharply by a zone of deep purple or chocolate color. Several observers[3,13] found bacteria, notably streptococci, in the thrombus, but the majority have reported sterile cultures. The thrombotic or infarcted area, either segmental or total, usually involves the entire parenchyma with necrosis and loss of the renal architecture. Occasionally there is an extension of the thrombosis to the opposite kidney, down the vena cava to the iliac, pelvic, spermatic, or ovarian veins, and upward to the pulmonary vascular tree and even the

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cerebral sinuses.[3] McClelland and Hughes[4] reported that in their series the uninvolved kidney revealed lower nephron nephrosis.

SYMPTOMS AND SIGNS

The clinical manifestations of the disease, as mentioned previously, usually have been prefaced by or are concomitant with an acute infection. It is interesting to note that in the author's case no evidence of infection could be found. In most cases, diarrhea associated with vomiting has been noted and has led to dehydration and peripheral collapse. Gross hematuria appears and the development of a tender, somewhat tense mass is found in the flank. Occasionally gross hematuria is not present, and because of the overwhelming sepsis or critical condition of the infant, involvement of the urinary system may be masked or overlooked. Even in the absence of a palpable tender kidney, the demonstration of albumin, pus, and blood in the urine should arouse more than a passing suspicion.[3] Anuria or oliguria, acidosis, and uremia are present in varying degree. Death usually occurs in a few days.

Excretory or retrograde urography will verify the diagnosis. Campbell and Matthews[7] advised doing both procedures. The characteristic findings in excretory urography are a blotting-out or absence of visualization of dye on the affected side when compared with the normal outline on the unaffected side. However, visualization of the urinary tract by intravenous pyelography in the newborn infant is notable for its inadequacies, and may be valueless. Retrograde pyelography, although it was not done in this case, is probably the most useful procedure for diagnostic purposes. It will reveal isolateral bleeding or diminished function on the affected side. The ureterogram is normal with an associated compression or blotting out of the pelvic markings. With an increased amount of material injected into the ureter of the affected side, Campbell[3] described an irregular diffusion of the media throughout the thrombosed organ, as though the injection had been made into a mass of mush, with no true pelvis outline demonstrated.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis should include adenomyosarcoma, polycystic kidney, hydronephrosis, acute pyelonephritis, renal or perinephritic abscess, and renal dysplasia with cystic mass. Retrograde pyelography should establish the diagnosis.

TREATMENT

Correction of shock, hydration, attention to electrolyte balance, and blood transfusion, if necessary, are of utmost importance in preparing the patient for surgery. Immediately upon verification of the diagnosis following urography studies, the patient should be considered to be in an emergency status and preoperative preparation, followed immediately by surgical inter-

vention, should be accomplished as quickly as possible. Nephrectomy of the thrombosed or infarcted kidney is performed. Abeshouse[5] emphasized that the surgeon should be certain to include the intravenous clot when the renal vein is ligated, to prevent extension of the thrombosis to the contralateral kidney. Rough and unnecessary handling of the renal pedicle should be avoided.

Postoperatively, the judicious use of whole blood, fluids, electrolytes, and antibiotics, as well as maintenance of an adequate caloric and protein intake, enhances the likelihood of successful management.

If both kidneys are involved, one can only offer supportive measures for a seemingly hopeless cause. Only one case of apparent bilateral renal vein thrombosis that survived has been reported.[6] One might consider employing anticoagulant therapy in such cases, in an effort to hinder advancement of parenchymal damage as well as to discourage development of thrombotic phenomena elsewhere. If surgery is contemplated, however, the use of anticoagulant measures prior to surgical intervention should be discouraged.

SUMMARY

A case of unilateral renal vein thrombosis that recovered following nephrectomy is presented. Renal vein thrombosis should be suspected in all infants presenting the clinical picture of infection, dehydration, hematuria, and palpable tender mass in the flank. Prompt retrograde or excretory urographic studies are imperative. Immediate nephrectomy is mandatory following preoperative correction of the dehydration and electrolyte imbalance. The correct preoperative diagnosis of unilateral renal vein thrombosis is of less importance than the expeditious initiation of the above-mentioned procedures.

REFERENCES

1. Avery, M. E.; Oppenheimer, E. H.; and Gordon, H. H.: Renal-vein thrombosis in newborn infants of diabetic mothers; report of 2 cases. *New England J. Med.* 256: 1134-1138, June 13, 1957.

2. Barenberg, L. H.; Greenstein, N. M.; Levy, W.; and Rosenbluth, S. B.: Renal thrombosis with infarction complicating diarrhea of newborn; summary of 5 cases. *Am. J. Dis. Child.* 62: 362-372, Aug. 1941.

3. Campbell, M. F.: *Pediatric Urology.* The Macmillan Company, New York, N. Y., 1937. Vol. I, p. 475.

4. McClelland, C. Q., and Hughes, J. P.: Thrombosis of renal vein in infants. *J. Pediat.* 36: 214-227, Feb. 1950.

5. Abeshouse, B. S.: Thrombosis and thrombophlebitis of renal veins. *Urol. & Cutan. Rev.* 49: 661-675, Nov. 1945.

6. Fallon, M. L.: Renal venous thrombosis in newborn. *Arch. Dis. Childhood.* 24: 125-128, June 1949.

7. Campbell, M. F., and Matthews, W. F.: Renal thrombosis in infancy; report of 2 cases in male infants urologically examined and cured by nephrectomy at 13 and 33 days of age. *J. Pediat.* 20: 604-615, May 1942.

8. Sandblom, P.: Renal thrombosis with infarction in newborn; two different forms. *Acta paediat.* 35: 160-167, 1948.

9. Morison, J. E.: Renal venous thrombosis and infarction in newborn. *Arch. Dis. Childhood* 20: 129-134, Sept. 1945.

10. Hepler, A. B.: Thrombosis of renal veins. *J. Urol.* 31: 527-546, Apr. 1934.

11. Zuelzer, W. W., and others: Circulatory diseases of kidneys in infancy and childhood. *A. M. A. Am. J. Dis. Child.* 81: 1-46, Jan. 1951.

12. Warren, H.; Birdsong, M.; Kelley, R. A.; Renal vein thrombosis in infants. *J. A. M. A.* 152: 700-701, June 20, 1953.

13. Kobernick, S. D.; Moore, J. R.; and Wiglesworth, F. W.: Thrombosis of renal veins with massive hemorrhagic infarction of kidneys in childhood. *Am. J. Path.* 27: 435-453, May-June 1951.

DIAGNOSIS OF EXPEDIENCY

"The appendix at birth has a wide lumen, and, as the best insurance against obstruction and development of acute inflammation, this feature explains why acute appendicitis is so rare in the neonate. Within two years, however, the submucosal lymphoid tissue has developed to a great degree, reducing luminal diameter and creating a cause for the sudden increase in the incidence of acute appendicitis. The opportunity for subclinical inflammation and fibrosis begins immediately. That both actual infection of the appendiceal wall and secondary inflammatory response within the lymphoid tissue to circulating noxious substances are common can be readily agreed upon; that the resulting intramural fibrosis cannot later be used as an explanation for clinical illness has been more difficult to accept. The pressures the clinician feels in this regard have to do with his natural compulsion to end up with an organic diagnosis, if that is at all possible. 'Chronic appendicitis,' like gastritis, prolapse of the gastric mucosa, and many others, becomes a diagnosis of enforced expediency."

—EDDY D. PALMER, M. D.
in *American Journal of Digestive Diseases*
p. 594-595, Oct. 1957

Heat Stroke: A Review of Three Cases

WILLIAM R. SCHILLHAMMER, *Major, MC, USA*
ROBERT L. MASSONNEAU, *Lieutenant Colonel, MC, USA*

HEAT STROKE is a state of hyperpyrexia associated with cessation of the body sweating mechanism. Prolonged excessive environmental heat is the principal etiologic factor. Humidity, wind velocity, acclimatization, and general physical condition, are other important contributory factors.[1] This condition is one of the few true medical emergencies for which treatment must be immediate to prevent death. The following patients were treated at this hospital during the summer of 1957, and have recovered from the effects of their sunstroke. We believe them to be of special interest because of the duration of their coma and the apparent dramatic efficacy of the treatment administered.

CASE REPORTS

Case 1. A 24-year-old white man, a basic trainee with 22 days' military service was admitted to this hospital, on 6 July 1957 at 1400 hours because of sudden loss of consciousness that had occurred one half hour previously. He had been on the rifle range from 0600 to 1200 hours that day, at which time the environmental temperature was in excess of 100°F. He marched back to his company area and collapsed shortly after. Cold sponges were applied and he was evacuated to the hospital emergency ward.

Physical examination. The patient was a comatose, well-developed, obese white man in acute distress. His respirations were Cheyne-Stokes in type; his temperature was 107.8°F (rectally); pulse rate, 180; blood pressure, 120/80 mm Hg. The remainder of the physical examination was within normal limits, except for absence of sweating and hot, dry skin.

Laboratory studies. Admission laboratory data revealed a white blood cell count of 17,000 per μl with 80 per cent neutrophils. Hemoglobin was normal. Urinalysis revealed 1 plus glycosuria and albuminuria. The electrocardiogram was normal. Subsequent checks of these findings revealed no abnormalities. Results of electrolyte studies were normal. The serologic test for syphilis was negative and roentgenogram of the chest, taken later, was normal.

Course in hospital. On arrival at the emergency ward, the patient was immediately immersed in a bathtub of water containing ice chips

From U. S. Army Hospital, Fort Hood, Tex.

9. Morison, J. E.: Renal venous thrombosis and infarction in newborn. *Arch. Dis. Childhood* 20: 129-134, Sept. 1945.

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Course in hospital. On arrival at the emergency ward, the patient was immediately immersed in a bathtub of water containing ice chips

From U. S. Army Hospital, Fort Hood, Tex.

and his temperature rapidly reduced. He was removed from the ice bath when his temperature was down to 100°F and was given 100 mg of hydrocortisone intravenously in dextrose and physiologic saline solution during three hours after admission, followed by 100 mg of cortisone acetate intramuscularly every eight hours. At the time of removal from the ice bath his blood pressure was unobtainable and it was necessary to institute antishock therapy. He was given a solution containing Levophed (brand of *l*-norepinephrine) intravenously at a rate appropriate to maintain his blood pressure within normal limits. The foot of his bed was elevated and oxygen was administered by nasal catheter. It was necessary to support him by this means for the next 12 hours, by which time the blood pressure had stabilized and Levophed was discontinued. Thorazine Hydrochloride (brand of chlorpromazine hydrochloride), 50 mg intramuscularly every four hours for two doses, was given with apparent effectiveness in reducing the extreme rigidity and struggling behavior which the patient exhibited. He remained comatose and unresponsive, however, and then displayed what appeared to be a typical picture of decerebrate rigidity. He required frequent nasopharyngeal suction to prevent obstruction, but tracheotomy was not believed to be necessary. Approximately 30 hours after admission he began to respond to strong stimuli and occasionally muttered unintelligible responses. By the morning of 8 July, after seeming to ascend through a gamut of basic neurologic states, he was able to converse rationally and had good recall of events. On 9 July it was noted that his scleras were icteric. Serum bilirubin at that time was 23.7 mg per 100 ml, with 9.1 direct reacting and 14.6 mg per 100 ml indirect reacting. Thymol turbidity was 4.4 units. Cephalin cholesterol flocculation was one plus, and serum cholesterol 164 mg per 100 ml with an esterified fraction of 80 per cent. Prothrombin time was 100 per cent and the SGO-T was 325 units. Plasma hemoglobin and red cell fragility studies were normal. The liver was palpated 2 fingersbreadth below the costal margin and was slightly tender. On 15 July, thymol turbidity was 6.2 units and 2-hour urinary urobilinogen was 1.2 units. The other tests were normal except for serum bilirubin of 14.2 mg per 100 ml with 8.5 indirect and 5.7 direct.

During the next month of hospitalization the jaundice gradually disappeared so that the patient had no residuals of his hepatitis. During this month, however, he showed various neurologic signs and symptoms. At times he had a positive Romberg's sign and at times abnormal reflexes. He also showed extrapyramidal signs with tremor of the hands and head and spasticity of the extremities. He walked unsteadily for a few days after regaining consciousness, but all these findings gradually improved.

The patient was given 30 days' convalescent leave from 20 August to 19 September. On return he complained regularly of headaches, dizziness, pain in the right foot, with difficulty in walking and other

symptoms which were very difficult to evaluate because of lack of supporting physical findings. Orthopedic and psychiatric consultations failed to disclose any other significant evidence of organic residuals. It was generally agreed that the patient had a passive aggressive personality pattern and was poorly motivated for further military service. Because a long-term medical and psychiatric evaluation seemed necessary, he was transferred to an Army General Hospital and eventually was separated.

Case 2. A 24-year-old white man, a basic trainee on active duty 32 days, was admitted to this hospital on 6 July 1957 at 1930 hours in a comatose state. The patient had been on the firing range from 0800 to 1600 hours the day of admission, then marched back to his company area. At 1930 hours he suddenly collapsed and was brought to the emergency room. His skin was hot and dry, and he was in shock. At the time of admission, rectal temperature was 108°F, pulse rate 180; respirations rapid, and blood pressure unobtainable. Physical examination was not otherwise remarkable.

Laboratory studies. Admission laboratory tests showed urinalysis, serum electrolytes, and chest roentgenogram to be within normal limits. An electrocardiogram was normal.

Course in hospital. The patient was immediately placed in a tub of ice water. He became combative and disoriented. By 2000 hours his temperature had fallen to 101°F, and the patient was removed from the ice bath and placed in bed. At 2005 hours his temperature was 97°F. He was extremely spastic and unresponsive, and assumed a fetal position from which he could not be moved.

No further temperature elevation occurred during the remainder of the hospital course. The patient was given 50 mg of Thorazine intramusuclarly and his spasticity decreased considerably. Intravenous administration of a solution of 5 per cent dextrose in saline containing 100 mg of hydrocortisone was instituted. He was given 100 mg of cortisone acetate intramuscularly every six hours for the next five days. He remained completely unresponsive, and at approximately 2245 hours his blood pressure, which had returned to a level of 100/70 mm Hg, suddenly fell markedly and it was necessary to give Levophed intravenously at a rate appropriate to control shock. By 2345 hours the patient had become flaccid and areflexic. His blood pressure was controlled at levels of approximately 100/70 mm Hg by slow Levophed drip.

About five hours after admission the patient suddenly became responsive, sat up, and asked for the doctor. He answered questions rationally and his neurologic examination at this time was normal. By the morning of 7 July 1957, it was possible to discontinue intravenous medication and he was able to take fluids by mouth. He progressively improved, was completely co-operative, but had some persistent generalized weakness for several days. He was allowed to be ambulatory and went on a week-end pass on 12 July 1957. His recovery was complete without residuals of physical or neurologic

damage and he was discharged to temporary limited (avoidance of prolonged exposure to sun) duty on 18 July 1957. He did very well on his return to duty, but eventually was discharged from the service for administrative reasons.

Case 3. A 26-year-old man of Mexican extraction, with 24 days of military service, was brought by ambulance to the emergency room of this hospital, Fort Hood, Tex., at approximately 1430 hours on 30 July 1957. He had been on the rifle range on the day of admission when he told someone he was not feeling well. Because of his appearance he was showered with cold water and immediately evacuated by jeep to his company area, where he collapsed in the orderly room. On arrival at the hospital he was comatose and his skin was hot and dry. He was cooled with ice chips and water and was sent to the emergency ward.

Physical examination. The patient was a well-developed, moderately obese Mexican male, who was comatose. His respirations were labored and rapid. His temperature was 109°F rectally; pulse rate 144; blood pressure at that time was unobtainable. The physical examination was not otherwise remarkable.

Laboratory studies. Urinalysis, hemogram, chest roentgenogram, and serum electrolyte studies were within normal limits. The serologic test for syphilis was negative.

Course in hospital. At 1445 hours he was placed in a tub of ice water. His temperature fell to 104°F at 1500 hours, and to 100°F at 1515 hours. At 1520 hours the patient was moved from the tub to his bed and an intravenous solution of 5 per cent dextrose and saline containing 100 mg of hydrocortisone was administered slowly. Cortisone acetate, 100 mg intramuscularly, also was started, and was continued every eight hours. He was extremely combative and still stuporous, and was given 50 mg of Thorazine intramuscularly, which served to quiet him somewhat.

By 1530 hours, the patient was responding to painful stimuli. His gag reflex was present, but he was resistive and his extremities were extremely spastic. Blood pressure was 110/70 mm Hg. At 1600 hours there was no change in his condition and he continued to exhibit outbursts of extreme psychomotor activity. He was given another 50 mg of Thorazine at this time, which again helped decrease his hyperactivity. He was given oxygen by nasal catheter during the entire period. At 2130 hours he was resting quietly and was responding to painful stimuli. There were no abnormal reflexes. At 2300 hours, about nine hours after onset of coma, he started talking, taking fluids, and seemed completely rational.

On the morning after admission, the patient was eating normally and felt well, except for mild generalized headache. Cortisone was continued orally in the dosage of 25 mg four times daily and discontinued on the fourth hospital day. The patient had no further signs or symptoms, and was discharged to temporarily limited duty on 12 August 1957.

DISCUSSION

The foregoing cases of severe sunstroke are presented to illustrate the effectiveness of treatment in this condition. These patients were comatose for 30, 5, and 9 hours, respectively, and yet have apparently recovered completely from the effects of their sunstroke. All authorities emphasize that proper management of a case of sunstroke demands return of the patient's temperature to normal as rapidly as possible. This can be accomplished by immersion of the entire body in a tub of ice water until the temperature is almost normal. (The tub and ice are kept ready for immediate use during the summer months.) Following the relief of hyperpyrexia, the patient must be adequately supported with proper nursing care, intravenous fluids in appropriate amounts, and control of blood pressure. Care must be taken that intravenous fluids not be administered too rapidly, for large quantities of fluid intravenously may precipitate pulmonary edema in these patients. For the management of hypotension during the critical phase, Levophed seems to be excellent. These patients also require frequent use of suction to avoid tracheal obstruction while in coma. Oxygen by nasal catheter serves to prevent hypoxia and to minimize the damage to the central nervous system that is caused by the hyperpyrexia and hypotension.

The condition of these patients was so serious that we used adrenocortical steroids, postulating that such severe trauma to the body must be associated with great adrenocortical stress and probably a significant degree of cortical failure or inadequacy. (This concept is neither proved nor controverted by pathologic findings. Malamud, Haymaker, and Custer[2] reported the gross and microscopic changes in the brain, liver, adrenals, kidneys, and other organs in a series of 125 autopsied cases. Hyperthermia *per se* was considered to be the proximate cause of the central nervous system damage, but findings in the parenchymal organs were of nonspecific etiology, were rare in cases of short survival, and could well have been secondary to hypoxia.) Thus, the use of exogenous steroids is based on the hypothesis of acute, severe, functional adrenal depletion due to stress. We gained the distinct impression that the adrenal steroids were beneficial and possibly lifesaving in these patients. Sampson and Yuen[3] and Waugh[4] have previously described the use of steroids in such patients.

Jaundice, such as that noted in case 1, has been described previously[5-7] and attributed to hepatocellular damage, presumably due to hypoxia; possibly also to hyperthermia.[2] The clinical and laboratory findings did not support such considerations as acute hemolysis and Thorazine hypersensitivity as causes of the jaundice.

Electrocardiographic and electroencephalographic abnormalities have been reported.[2,5] These examinations gave normal findings in case 1, and the electrocardiogram was normal in case 2.

The exact mechanism of heat stroke is unknown. However, there is considerable information which indicates that the problem results from fatigue of the heat regulation center.[1,2,6-11] It has been shown that prolonged exposure to heat with continuous perspiration gradually decreases the efficiency of the heat regulation center and the sweating mechanism of the body. If a person is able to obtain freedom from such an environment at intervals during the day, such as in an air-conditioned room, heat stroke is not likely to occur. In the summer at Fort Hood, Tex., the temperature occasionally rises as high as 105°F and frequently does not drop below 80°F at night. The average humidity during the month of July 1957 was 79 per cent. The barracks were not air-conditioned and hence the patients perspired 24 hours daily. Although heat precautions normally are taken, and the commanders and noncommissioned officers are instructed in the cause and nature of heat injuries, all troops have not been adequately protected at all times.

The patients described were placed on the critically ill list and a fatal outcome was anticipated. Early and adequate treatment, including a vasopressor agent and adrenal steroids intravenously, apparently enabled them to recover completely. We report their cases to encourage vigorous therapy and a more optimistic prognosis in patients admitted for this condition in the future.

REFERENCES

1. Schickele, E.: Environment and fatal heat stroke; analysis of 157 cases occurring in Army in U. S. during World War II. *Mil. Surgeon* 100: 235-256, Mar. 1947.

2. Malamud, N.; Haymaker, W.; and Custer, R. P.: Heat stroke; clinico-pathologic study of 125 fatal cases. *Mil. Surgeon* 99: 397-449, Nov. 1946.

3. Sampson, H. A., and Yuen, L.: Use of ACTH in heat sickness. *New York J. Med.* 54: 3420, Dec. 15, 1954.

4. Waugh, W. H.: Cortisone and treatment of heat stroke. *Ann. Int. Med.* 41: 841-843, Oct. 1954.

5. Logue, R. B., and Hanson, J. F.: Electrocardiographic changes following heat stroke; report of case. *Ann. Int. Med.* 24: 123-127, Jan. 1946.

6. Wright, D. O.; Reppart, L. B.; and Cuttino, J. T.: Purpuric manifestations of heatstroke; studies of prothrombin and platelets in 12 cases. *Arch. Int. Med.* 77: 27-36, Jan. 1946.

7. Saffert, C. A.: Heat hyperpyrexia; report of extreme case surviving for 8½ days. *Minnesota Med.* 20: 106-108, Feb. 1937.

8. Daily, W. M., and Harrison, T. R.: Study of mechanism and treatment of experimental heat pyrexia. *Am. J. Med. Sc.* 215: 42-55, Jan. 1948.

9. Austin, M. G., and Berry, J. W. (St. Louis): Observations on 100 cases of heat stroke. *J. A. M. A.* 161: 1525-1529, Aug. 18, 1956.

10. Cecil, R. L.; Loeb, R. F.; and others (editors): *A Textbook of Medicine.* 9th edition. W. B. Saunders Co., Philadelphia, Pa., 1955. p. 518.

11. *The Etiology, Prevention, Diagnosis and Treatment of Adverse Effects of Heat.* TB. MED. 175/NAVMED P-5052.5/AFP 160-4-1A, 7 August 1957 and Changes No. 1, 26 Nov. 1957. Departments of the Army, the Navy, and the Air Force, Washington 25, D. C.

Fetus Papyraceous and a Microcephalic Infant

SAM P. PATTERSON, *Captain, MC, USA*

FETUS PAPYRACEOUS is a rather rare condition in which one of twin fetuses has died and been pressed flat against the uterine wall by the living twin. Since Kindred[1] reviewed the literature in 1944 and summarized 150 cases, a number of additional cases have been reported.[2-11] The present case was not complicated by polyhydramnios, as many are; and nothing could be determined regarding the umbilical cord of the fetus or its relation to the placenta. In most cases, the compressed fetus is in the fundus, but here it was in the lower part of the uterus and delivered first. As in many other cases, the viable infant was abnormal.

CASE REPORT

A 20-year-old Caucasian woman was first seen in the prenatal clinic of this hospital on 27 February 1957. Her last normal menstrual period had been 12 October 1956, and her estimated date of confinement was 19 July 1957. She was para 2, gravida 3.

Past history on her initial visit revealed that she had had a kidney infection with one of her previous pregnancies and had had swelling of the feet and ankles with both pregnancies. Her first pregnancy terminated with delivery of a normal male infant 17 November 1954. She delivered a normal female infant 26 June 1956. There was no known viral infection during her present pregnancy.

On her initial clinic visit in February she weighed 136½ lb (usual weight, 120). She was five feet tall. Her blood was type AB, Rh positive, with a hemoglobin of 12.4 grams per 100 ml. Her urine was negative for albumin and sugar on that visit and on each subsequent visit. The serologic test for syphilis was negative. On 18 June her hemoglobin was 12 grams per 100 ml. Her prenatal course was complicated by an excessive weight gain of 28 3/4 lb. At approximately seven months gestation, the patient developed pitting edema of the feet and ankles. She was placed on a 1,200-calorie, low-salt, high-protein diet, and was given Diamox (brand of acetazolamide). The edema subsided for a few weeks but then recurred and persisted until delivery. During this period the patient's blood pressure remained normal and her urine negative for albumin. At no time during the prenatal

From U. S. Army Hospital, Fort Bragg, N. C.

course was it thought that she had twins or that she had any evidence of polyhydramnios.

She was admitted to the labor suite 24 July in active labor with the vertex presenting; her blood pressure was 116/80 mm Hg, and the fetal heart tones were normal in the left lower quadrant. Labor progressed rapidly, and she was taken to the delivery room two hours after admission. Rectal examination at that time revealed the cervix to be dilated to a rim. As the fetal head came down quickly from a zero station to the perineum, a macerated fetus papyraceous was

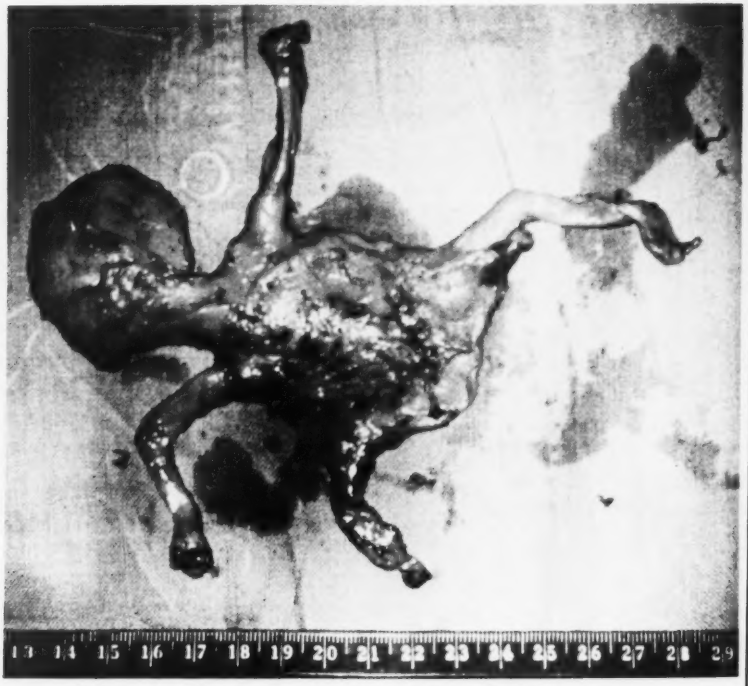

Figure 1. Macerated fetus papyraceous.

suddenly expelled from the vagina. It had evidently been wedged between the viable infant's head and the walls of the pelvis during descent. The viable twin was then delivered from the right occipito-anterior position over a midline episiotomy. The fetus papyraceous was about 15 cm in length and 1.5 cm thick (fig. 1). It was very macerated but well preserved. The sex was not determined. The second fetus weighed 8 pounds, 6 ounces, and was a male (fig. 2). This infant was microcephalic. The placenta was large, but no weight was obtained. It appeared normal with no areas of infarction. Alongside one portion of the membranes was a distorted area with several adherent fragments of what may have been the membranes of the fetus papy-

raceous. It could not be determined where the umbilical cord of this fetus might have entered the placenta.

Figure 2. Fetus papyraceous with large microcephalic twin. This photograph, unfortunately, does not demonstrate the microcephaly.

The mother's postpartum course was uneventful. The infant took its formula well and was discharged with the parents, who were warned of the ultimate prognosis.

REFERENCES

1. Kindred, J. E.: Twin pregnancies with one twin blighted; report of two cases with comparative study of cases in literature. *Am. J. Obst. & Gynec.* 48: 642-682, Nov. 1944.

2. Mulliken, D. K.: Fetus compressus; case report. *Am. J. Obst. & Gynec.* 71: 223-224, Jan. 1956.

3. Southern, E. M.: Case of foetus papyraceus. *Brit. M. J.* No. 4966: 556, Mar. 10, 1956.

4. Forman, R. C.: Twin pregnancy with one twin blighted. *Am. J. Obst. & Gynec.* 72: 1180-1181, Dec. 1956.

5. Sabath, D. J.; Kvitek, L. C.; and Baugher, A. H.: Fetus papyraceous in binovular twin pregnancy. *Am. J. Obst. & Gynec.* 56: 989-990, Nov. 1948.

6. Arends, N. W.; Dressler, O.; Thornton, W. H.; and Kester, G. E.: Instance of fetus papyraceus probably due to trauma. *J. Am. Osteopath. Ass.* 50: 611-613, Aug. 1951.

7. Posner, A. C., and Klein, M. A.: Fetus papyraceus; recognition and significance. *Obst. & Gynec.* 3: 106-110, Jan. 1954.

8. Cornacchione, M.; Gotlib, M. H.; Huse, W.; and Traube, J. I.: Twins: one macerated, the other near term and normal; case report. *J. Indiana M. A.* 43: 1207-1210, Dec. 1950.

9. Roos, F. J.; Roter, A. M.; and Molina, F. A.: Case of triplets including anomalous twins and fetus compressus. *Am. J. Obst. & Gynec.* 73: 1342-1345, June 1957.

10. Sinclair, R. St. C.: Foetus papyraceus. *South African M. J.* 26: 69-71, Jan. 26, 1952.

11. Mira, W. J. D.: Foetus papyraceus: unusual case. *M. J. Australia* 1: 591-593, Apr. 25, 1953.

NO BED OF ROSES

"Obstetrics is thought by many to be the most stagnant field in medicine. It is felt that nothing new ever happens and that it is a 'real bed of roses.' These statements are far from any truth. We are constantly striving for and achieving a lower fetal and maternal mortality. We are improving our antenatal care. We are conducting our labors and deliveries with better and safer analgesics and anesthetics. We are having more normal puerperiums. Finally, we are getting healthier mothers and babies. If one does obstetrics long enough he will find that the 'bed of roses' is padded with many obstetrical emergencies, such as sudden, severe, hemorrhages, shocks of every description, diseases of the parturient mother of every kind, and difficult deliveries due to abnormal presentations."

—ROBERT W. ROSS, M. D.
in *Journal of the Arkansas Medical Society*
p. 196, Oct. 1957

Tracheoesophageal Fistula Without Atresia of Esophagus

Diagnosis and Repair in the Newborn

PHILIP L. NOVA, *Captain, MC, USN*

CONGENITAL tracheoesophageal fistula without other anomalies of the esophagus is relatively rare. In a combined series of 201 cases of congenital tracheoesophageal anomalies recently reported by Haight,[1,2] tracheoesophageal fistula alone was found in 3 per cent. In 1954 Ware and Cross[3] found 26 cases of this anomaly in the English literature, and added another one.

In newborn infants, the symptoms of tracheoesophageal fistula without other associated anomalies of the esophagus may not be too severe, and consequently may remain unsuspected. The life expectancy of most untreated cases is relatively brief, averaging from a few days to a few weeks, depending on the severity of the pneumonitis that develops.

The diagnosis, even when suspected, has presented some difficulties, but if certain measures are carried out during the roentgenologic examination with contrast medium, the diagnosis may be readily established. Once the diagnosis is made, surgical correction should be carried out as soon as possible.

CASE REPORT

Case 1. A white male infant, the second child of a 21-year-old mother, was born at this hospital on 18 August 1956. The prenatal course and delivery had been uncomplicated. His weight at birth was 8 3/4 lb, his cry was lusty, no resuscitative measures were used, and his general appearance was good. There was a considerable amount of mucus in the mouth, nose, and upper respiratory passages, and loud, coarse rales were heard throughout both lung fields. There was no cyanosis.

During the first 24 hours of life the baby continued to have increased mucous secretions in the pharynx, although no fluids were given. On the second day he received a small amount of glucose solution by mouth. This promptly produced an episode of coughing, choking, and cyanosis. A small polyethylene tube was readily passed into the

From U. S. Naval Hospital, Portsmouth, Va.

stomach, confirming patency of the esophagus, and was left in the stomach for administration of glucose solution.

The preliminary roentgenographic examination revealed clear lung fields, no gross cardiac abnormalities, and an increased amount of air in the intestines (fig. 1). A small quantity of iodized oil was placed in the baby's mouth, and roentgenograms taken in the conventional

Figure 1. Roentgenogram taken after the first 24 hours of life, showing clear lung fields, no gross cardiac abnormalities, and an increased amount of air in the intestines.

manner outlined the esophagus and stomach as well as the trachea and bronchi (fig. 2). No communication was demonstrated between the esophagus and trachea.

After the infant was returned to the nursery, several additional attempts were made to give him glucose solution by mouth. Each time this caused coughing, choking, and cyanosis. Because tracheoesophageal fistula was suspected, formula feedings were not given. The baby was kept hydrated by giving the glucose solution in small quantities through

the tube placed in his stomach. He tolerated this without difficulty, particularly when he was held in the upright position.

Additional roentgenographic studies were carried out on the third day. A small rubber catheter was placed in the stomach, and the baby held in the prone position on the x-ray table. Under fluoroscopic guid-

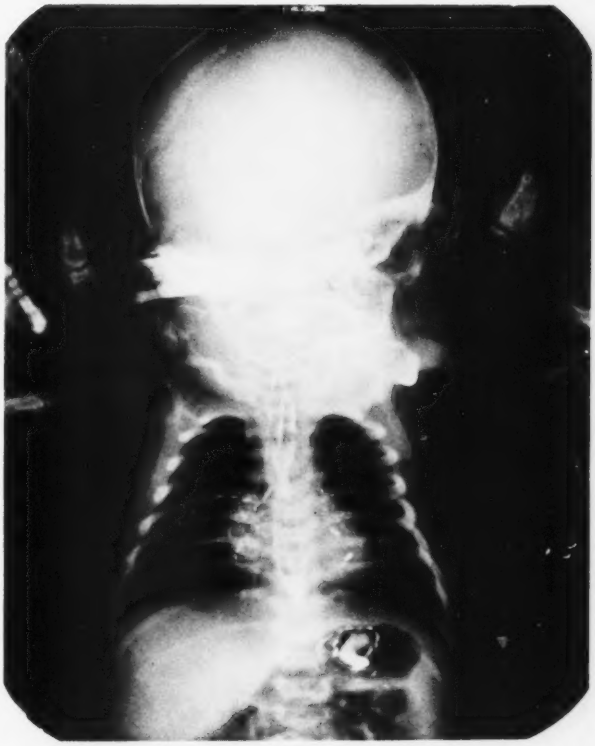

Figure 2. Roentgenogram taken in the conventional manner with the baby in the supine position after a small amount of iodized oil had been placed in his mouth. The esophagus, trachea, and bronchi are well outlined, but the fistula was not visualized.

ance, a small quantity of Lipiodol (brand of iodized poppy seed oil) was injected into the catheter as it was slowly withdrawn along the course of the esophagus. When the tip of the catheter reached the approximate level of the bifurcation of the trachea, the contrast medium was seen to flow into the dependent trachea. Spot films taken at that point revealed a communication between the esophagus and trachea about 2 cm above the carina. The fistula was diagonally placed with the tracheal opening slightly above the opening in the esophagus (fig. 3).

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Operation and repair of the fistula was performed on the following day. A right transpleural approach was used under endotracheal anesthesia. The tracheoesophageal fistula was found on the right lateral wall of the trachea 2 cm above the bifurcation, it was about ½ cm in length, and 2 to 3 mm in diameter. Its tracheal connection was elliptic in shape. To facilitate closure it was transected with a wider

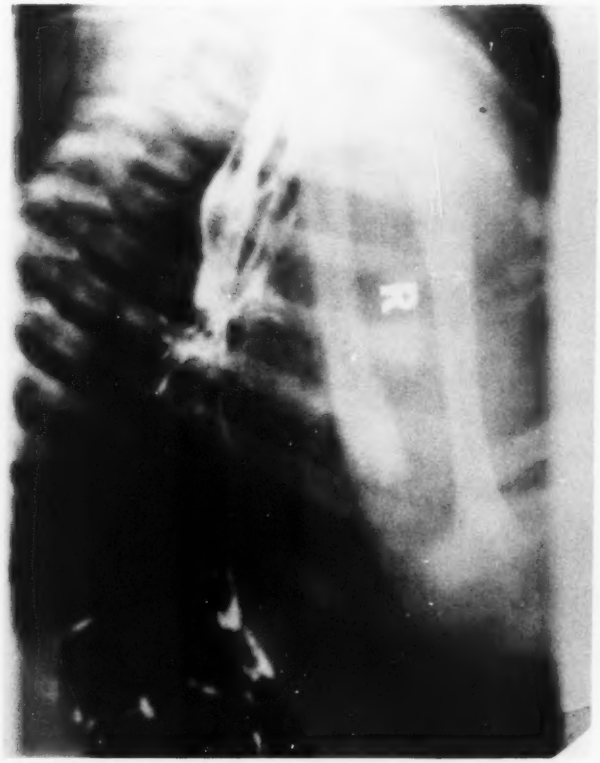


Figure 3. Roentgenogram taken with the baby held in the prone position. Iodized oil was injected through the catheter in the esophagus. The fistula is clearly demonstrated.

cuff on the tracheal side. The opening in the trachea was closed with a single row of interrupted sutures of No. 00000 atraumatic silk and the suture line was covered with a small flap of mediastinal pleura. The opening in the esophagus was closed in two layers using interrupted sutures of No. 00000 atraumatic silk.

The baby's subsequent immediate postoperative course and convalescence were uncomplicated. A diluted formula feeding was given orally, beginning on the third postoperative day. He was discharged

from the hospital on the 15th postoperative day, all feedings being taken without symptoms.

He was seen at periodic intervals in the outpatient clinic and his growth and development continued normally. At 6 months of age his weight had increased to 14 lb 10 oz, and he continued to be free of symptoms.

DISCUSSION

The majority of congenital esophageal malformations consist of an atresia that usually is associated with a fistulous communication between the trachea and lower esophageal segment. The various types which occur have been well documented. According to Vogt's[4] classification, these are:

Type I—Complete absence of the esophagus.

Type II—Atresia of the esophagus with an upper and a lower esophageal segment, each ending in a blind pouch.

Type III—Atresia of the esophagus with tracheoesophageal fistula, with fistula between (a) the upper segment and the trachea; (b) the lower segment and the trachea or a bronchus (91 per cent of the cases according to Haight[1]); and (c) both segments and the air passages.

A fourth type, in which there is tracheoesophageal fistula without atresia, was reported by Haight[1] to have been seen in 3 per cent of his combined series of cases.

In a case of suspected tracheoesophageal fistula without atresia, the roentgenologic diagnosis may not be apparent if the examination is carried out with the patient in the supine position. The importance of roentgenologic examination with the patient in the prone position in these cases has been stressed by Holt, Haight, and Hodges.[5] The prone position causes the fistula to be dependent in its relation to the esophagus, thereby allowing the iodized oil to enter the trachea by gravitation.

SUMMARY

Congenital tracheoesophageal fistula without an associated atresia of the esophagus is of rare occurrence. It has been observed in approximately 3 per cent of the cases of congenital esophageal anomalies. The diagnosis may not be apparent on roentgen examination with a contrast medium, unless examination is carried out with the patient in the prone position. The case of a newborn infant in whom a tracheoesophageal fistula was suspected, and demonstrated by roentgenograms and repaired successfully on the fourth day of life, is presented.

REFERENCES

1. Haight, C.: Some observations on esophageal atresia and tracheoesophageal fistulas of congenital origin. *J. Thoracic Surg.* 34: 141-172, Aug. 1957.

2. Haight, C.: Congenital tracheoesophageal fistula without esophageal atresia. *J. Thoracic Surg.* 17: 600-612, Oct. 1948.

3. Ware, G. W., and Cross, L. L.: Congenital tracheo-esophageal fistula without atresia of esophagus. *Pediatrics* 14: 254-258, Sept. 1954.

4. Vogt, E. C.: Congenital esophageal atresia. *Am. J. Roentgenol.* 22: 463-465, Nov. 1929.

5. Holt, J. F.; Haight, C.; and Hodges, F. J.: Congenital atresia of esophagus and tracheo-esophageal fistula. *Radiology* 47: 457-470, Nov. 1946.

A HOSPITAL "SPECIAL CARE UNIT"

"A special care unit is a segregated hospital area in which acutely ill patients are concentrated. The unit is specially equipped, has an augmented local supply, and an augmented staff. The function of such a unit is to provide special, intensive, and expert care for the acutely ill. Such a unit has been in operation at the Hitchcock Hospital in Hanover, New Hampshire, for the past two years, and has proven a valuable asset for the medical staff, the nurses, and the patients. The reasons for establishing such a unit are many. Primary is the problem of the increasing number of acutely ill in our hospitals. Advances in medical and surgical treatment are producing patients requiring special attention and preserving patients heretofore beyond salvage. These patients need intensive nursing care, yet the supply of nurses is short and unable to cope with the ever increasing load. It is obvious that more efficient utilization of personnel and concentration of effort is essential if we are to fulfill our obligations to these acutely ill patients."

—WILLIAM T. MOSENTHAL, M. D.
in *Journal of the Maine Medical Association*
p. 396, Nov. 1957

Lipoma of the Oral Cavity

BYRON G. McKIBBEN, *Colonel, MC, USA*
WALTER B. EIDBO, *Captain, MC, USA*

LIPOMAS may occur wherever fatty tissue is found, and occasionally are seen in the oral cavity.[1] In this location, they generally arise from the fat cells in the gingiva, or from adipose tissue in the buccal space, tongue, or floor of the mouth. Congenital lipomas of the cheek have been reported.[2] While deep-seated lipomas in other parts of the body have been known to undergo malignant change, those in the oral cavity apparently always remain benign.[3]

Lipomas are well-defined, soft tumors and produce no symptoms other than those due to their mass. They may be pedunculated or sessile, may be rounded or have variable formations with several lobes, and have been found bilateral. They are not resorbed like natural fat stores, which are removed when the person loses weight.[1,4]

In 1934, Geschickter[3] reported on a series of 622 lipoid tumors recorded in the files of the Surgical Pathological Laboratories of Johns Hopkins Hospital. His analysis showed that 460 of these tumors were benign lipomas, and that only three of them were located in the oral cavity.

At the Armed Forces Institute of Pathology, during the five-year period, 1953 to 1958, 1,768 primary tumors were recorded in the Registry of Oral Pathology. These were all types, both benign and malignant. Of the 1,768 tumors, 17 (less than 1 per cent) were lipomas.[5]

CASE REPORT

A 38-year-old Negro woman was first seen in the ear, nose, and throat clinic of this hospital, 21 November 1957, because of a mass in the right cheek. She stated that she had first noticed the mass four months previously and that it had increased slightly in size.

On physical examination, a discrete, soft, nontender, freely movable mass, measuring about 4 cm in its greatest diameter, was palpated in the right cheek, lateral to the mandible. The tumor caused the right

From William Beaumont Army Hospital, El Paso, Tex.

cheek to bulge about 1 cm, and with the mouth open the buccal surface also was seen to bulge. The neck was free of palpable nodes.

The patient was admitted to the hospital, 10 January 1958, and under anesthesia with 1 per cent infiltration of Xylocaine Hydrochloride (brand of lidocaine hydrochloride) an incision was made through the buccal mucous membrane, and the tumor—which was well encapsulated—was easily shelled out by blunt dissection. The fibrous capsule of the tumor appeared to be continuous with the fascia along the lateral side of the mandible. A rubber drain was inserted and the wound was closed with No. 0000 plain gut sutures.

The gross specimen was somewhat egg-shaped and measured 4 by 2.5 by 1.5 cm. The external surface was smooth, yellow in color, and was covered with a thin fibrous capsule. The cut surface was yellow, greasy, lobulated, and had the appearance of adipose tissue (fig. 1).

Figure 1. Appearance of the gross specimen.

Microscopic examination showed lobules of benign adult fat cells. A network of connective tissue separated the lobules and formed the capsule. Benign vascular structures were seen throughout the tumor mass (fig. 2).

Comment: It is interesting to note that a tumor of this size, located in the cheek, was not observed by the patient until 4 months prior to the time when she first sought medical attention. Lipomas are known to be slow-growing, and this one must have been large enough to cause a noticeable bulge and to be palpated for well over a year prior to the time of its detection by the patient.

SUMMARY

Lipomas of the oral cavity are not common. They are slow-growing and benign, and produce no symptoms other than those due to their mass. One case in a Negro woman is presented.

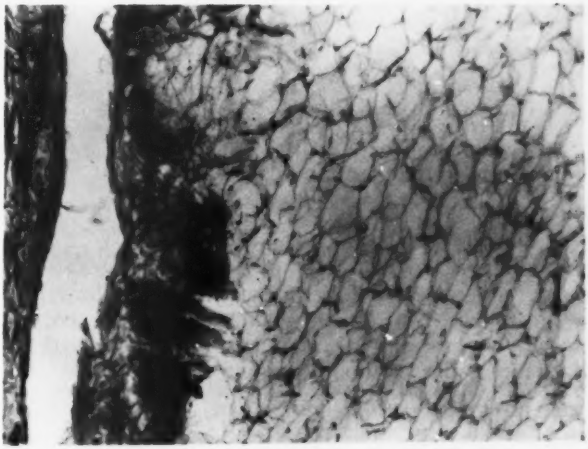


Figure 2. Photomicrograph showing connective tissue capsule to the left. Adult adipose cells are seen in the remainder of the section. (× 35)

REFERENCES

1. Ward, G. E., and Hendrick, J. W.: *Diagnosis and Treatment of Tumors of the Head and Neck.* Williams & Wilkins Co., Baltimore, Md., 1950. pp. 236-239.

2. Ransohoff, J.: Congenital lipomata of cheek. *Ann. Surg.* 65: 711, June 1917. Cited in reference 3.

3. Geschickter, C. F.: Lipoid tumors. *Am. J. Cancer* 21: 617-641, July 1934.

4. Thoma, K. H.: *Oral Pathology.* 4th edition. C. V. Mosby Co., St. Louis, Mo., 1954. pp. 1387-1390.

5. Bernier, J. L.: Personal communication.

WHY SEAT BELTS?

"No single safety device has been more effective in a crashing auto than the properly installed seat belt. On this the experts agree, especially in a market of high speed, power brakes, and trigger-happy driver feet."

—MEDICINE AT WORK
in *Journal of the American Medical Association*
p. 889, Oct. 27, 1956

Thymoma and Myasthenia Gravis

HAROLD COLLINGS, Jr., *Major, MC, USA*
WILLIAM R. SWEETMAN, *Major, MC, USAR*

THE association of thymoma and myasthenia gravis has been known since 1901. Sixteen per cent of a large series of patients with myasthenia gravis were found to have thymomas.[1] As many as 75 per cent of patients with thymoma have had associated myasthenia gravis.[2]

The causal relationship between thymoma and myasthenia gravis is not certain. The fact that myasthenia gravis may develop several years after the diagnosis of thymoma makes it unlikely that the thymoma is caused by the myasthenia gravis. That the thymoma causes the myasthenia gravis is an attractive possibility which is supported in many cases by remission or marked improvement of the disease following treatment of the thymoma. Some experimental evidence has been obtained that extracts of fetal whale thymus and of human thymomas have a myasthenia gravis-like effect on nerve-muscle preparations and on animals.[3] However, reports of cases in which myasthenia gravis developed after the removal of thymomas[4-6] have raised doubts that thymomas cause myasthenia gravis. The development of myasthenia gravis during the early postoperative period in some of these cases is not convincing evidence against such a causal relationship. In spite of apparently complete removal of a thymoma, there may be recurrence of thymoma;[7] and in only one of these reported cases of myasthenia gravis developing after the postoperative period has there been autopsy evidence that no recurrent tumor was present. (In that case there were some hyperplastic thymus remnants.[4]) These reports of myasthenia gravis developing after surgical removal of a thymoma do not disprove the idea that thymomas may cause myasthenia gravis. That both thymoma and myasthenia gravis are caused by a third unknown factor is a possibility.

The myasthenia gravis associated with thymoma usually is rapidly progressive and severe; treatment is a difficult problem. Such cases are relatively rare, and all should be reported so that eventually some conclusions may be drawn regarding proper management. Since 1947 two patients with thymoma and myas-

From Fitzsimons Army Hospital, Denver, Colo. Dr. Collings is now at Guthrie Clinic, Sayre, Pa.

thenia gravis have been seen at this hospital. The first case is taken from the hospital records; the second patient was seen and treated by the authors.

CASE REPORTS

Case 1. A 51-year-old man had been completely asymptomatic, but was transferred to this hospital on 31 March 1947 because of the finding of a mediastinal mass on a routine chest roentgenogram. Past history was not significant except for congenital syphilis which had been treated with bismuth, arsenicals, and artificial fever.

Physical examination was not remarkable. Roentgenograms of the chest revealed a sharply circumscribed tumor mass extending laterally and anteriorly from the left hilar region (figs. 1 and 2).

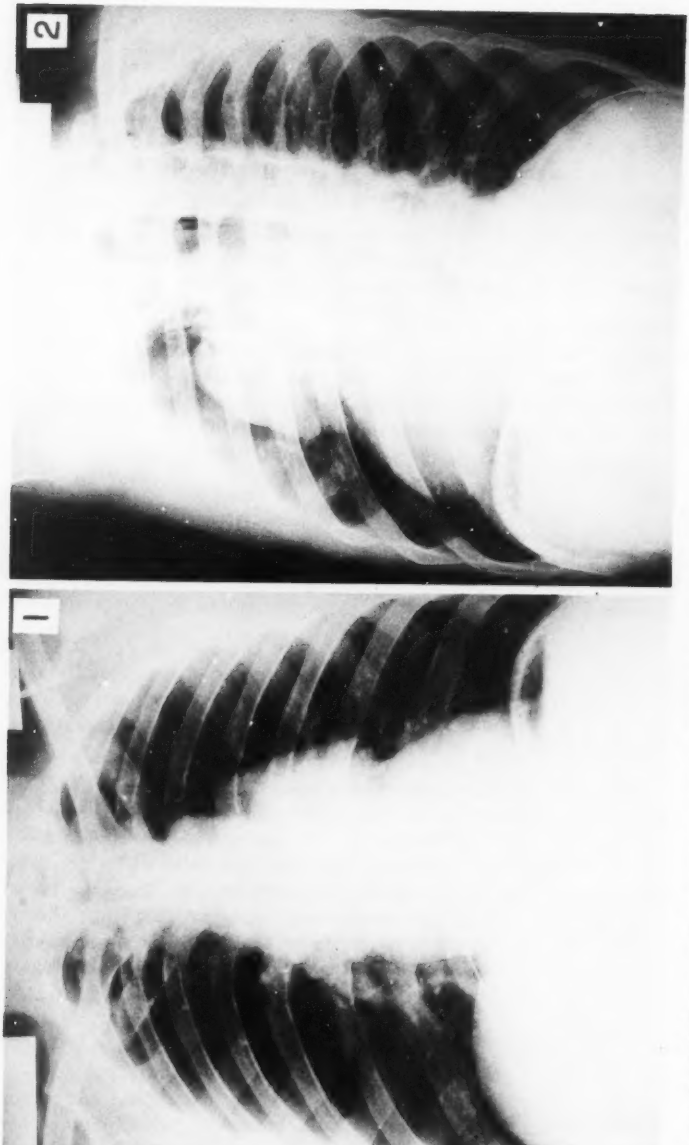
On 16 April a left thoracotomy was performed, and a tumor was found in the anterior mediastinum just anterior to and above the arch of the aorta. The tumor was very densely adherent to the pericardium and could not be removed. Microscopic examination of the biopsy specimen revealed that it was a thymoma.

The postoperative course was uneventful, and from 29 April to 23 May 1947 the patient received 2,200 r of radiation to each of two fields over the mid-chest anteriorly and posteriorly.

The patient was last seen at this hospital 2 July 1948, at which time he was asymptomatic and demonstrated no evidence of myasthenia gravis.

A letter dated 30 May 1949 stated that the patient recently had developed severe myasthenia gravis requiring large doses of Prostigmin (brand of neostigmine). Subsequent correspondence related that the patient died 9 June 1949 of respiratory paralysis, myasthenia gravis, and tumor of the thymus.

Case 2. A 39-year-old woman was admitted to this hospital for the first time on 4 October 1955. In August she had noted inability to chew, and a month later difficulty in swallowing; she had also noted difficulty in talking and weakness of her extremities. Activity increased all symptoms. After noting drooping of the left eyelid and vertical diplopia she was hospitalized at another hospital where a neostigmine test was positive for myasthenia gravis. She was started on small doses of neostigmine and transferred to this hospital. Examination revealed ptosis, vertical diplopia; severe weakness of masticator muscles; nasal voice; marked weakness of neck muscles; moderate weakness of muscles in the upper extremities, most marked in proximal muscles; and some weakness of hip girdle muscles. Radioactive iodine uptake studies revealed no evidence of hyperthyroidism. A postero-anterior roentgenogram of the chest was normal. Gradually increased neostigmine dosage resulted in decreasing muscle weakness, so that by 29 October the patient was fairly well controlled on daily intake of 33 tablets (15 mg per tablet) of neostigmine and was discharged from the hospital.



Figures 1 and 2 (case 1). Roentgenograms of the chest showing a tumor mass extending anteriorly and laterally from the left hilar region.

The patient was admitted to this hospital for the second time on 22 May 1956 with the history that her weakness had rapidly increased in spite of taking 30 tablets of neostigmine and 22 tablets (60 mg per tablet) of Mestinon (brand of pyridostigmin bromide) daily. She had been very weak most of the time and had had at least two episodes of acute respiratory distress. Roentgenograms (including a lateral view) of the chest revealed a nodular anterior mediastinal mass in the region of the ascending aorta (figs. 3 and 4).

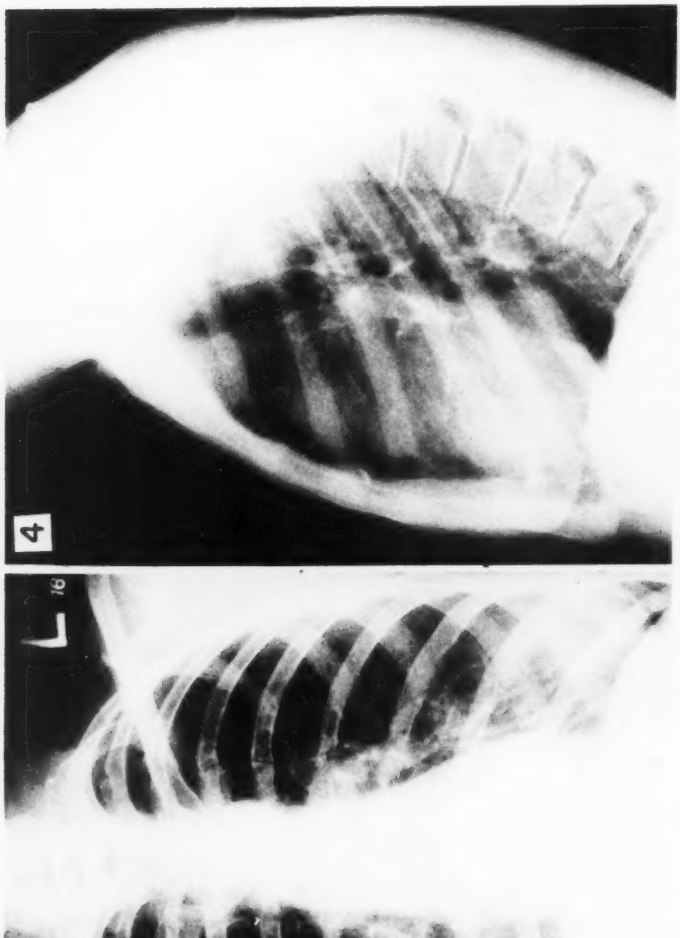
On 26 May the patient had the onset of progressive respiratory distress so that after several minutes respirations were minimal and cyanosis was present. The administration of Tensilon Chloride (brand of edrophonium) intravenously produced dramatic improvement in her condition, which was maintained with neostigmine administered intramuscularly. Oral medication was gradually increased to a total of 63 tablets of neostigmine and of Mestinon each day.

From 7 June to 14 July the patient was given irradiation therapy with a total dose of 3,994 r to the mediastinal tumor. During this time her neostigmine requirements decreased to 40 tablets daily. Roentgen studies indicated that the mediastinal mass had decreased about 50 per cent in size. The patient was discharged from the hospital for three weeks prior to surgery. By the time of the third admission on 14 August 1956 her neostigmine requirements had decreased to 26 tablets daily.

On 22 August a right thoracotomy was performed under gas-oxygen-ether and Pentothal (brand of thiopental sodium) anesthesia. A lobulated, egg-shaped, firm, movable tumor mass measuring 8 by 5 by 7 cm was found in the anterosuperior mediastinum overlying the arch of the aorta. It was adherent to the pericardium but seemed to be entirely encapsulated. The tumor was totally removed. Following thoracotomy, a tracheotomy was performed as a precautionary measure in the management of myasthenia during her postoperative period.

The patient was not given neostigmine on the morning of surgery, but was given 1.5 mg intramuscularly at the completion of the operation. She was observed carefully, but required no more for some 18 hours, at which time increasing weakness and apprehension appeared. She received 1 mg of neostigmine intramuscularly every two or three hours for the next week. One week postoperatively, oral medication was resumed. On 22 tablets of neostigmine daily, muscular twitching occurred, and that medication was gradually decreased to 15 tablets daily by 8 September.

The patient was discharged from the hospital 26 September, and returned to her factory job in October 1956. When last seen, on 23 April 1957, she no longer took neostigmine regularly (although once every few days she took 1/4 of 1 tablet) and had been asymptomatic. Examination at that time revealed only mild weakness of shoulder girdle muscles. Roentgenographic examination of the chest showed no recurrence of tumor. In April 1958 the patient wrote that she was doing well and was taking from 1 to 3 tablets of neostigmine each day.



Figures 3 and 4 (case 2). Roentgenograms of the chest showing an anterior mediastinal mass in the region of the ascending aorta.

DISCUSSION

The prognosis in patients with myasthenia gravis and untreated thymoma is very poor. Either the myasthenia gravis or the untreated thymoma represents sooner or later a serious threat to life.

Although it is generally agreed that thymectomy is of value in the treatment of progressively severe, generalized myasthenia gravis in young women without thymomas, results with surgical removal of thymomas in the presence of myasthenia gravis without prior irradiation therapy have been uniformly bad. Of nine such patients reported by Grob[8] from the Johns Hopkins Hospital, six died of myasthenia gravis. Schwab and Leland,[9] in reporting the series from the Massachusetts General Hospital, concluded that the results were no better with surgical intervention than without it. In the series of 39 patients with surgical treatment of thymoma reported by Eaton and Clagett[1] from the Mayo Clinic, there were 5 operative deaths and 14 subsequent deaths (all but one of myasthenia gravis); in only 13 was there symptomatic improvement. In a total of 11 patients reported by Keynes[10] in whom surgical treatment was undertaken without previous irradiation therapy, there were 3 operative deaths. Only 3 patients were living after two years, and only 1 was living at the time of Keynes' report.

No large series of patients treated only with irradiation therapy has been reported. Some reported cases, however, suggest that it does frequently bring about an improvement in the myasthenia gravis and the tumor.[10] In some reported cases, it seems likely that irradiation therapy of the thymoma delayed the onset of myasthenia gravis. This perhaps was true in case 1.

Although Keynes' results in the early patients treated only with thymectomy were very poor, the results were much better in 21 cases treated first with irradiation therapy followed by surgical intervention.[10] Four patients were well up to five years after surgery; 8 were much improved; 1 was unchanged; and 5 patients died. The results thus were gratifying in 12 out of 21 patients. It is possible that the development of improved surgical and anesthetic technics and more experience in the medical management of myasthenia gravis partially account for the difference in results in Keynes' two series of patients. It seems most likely, however, that irradiation therapy was at least partially responsible for the better results.

Every patient with myasthenia gravis should have roentgenographic examination (including lateral or oblique views) of the chest for evidence of thymoma. If such is found, it is believed the tumor should be treated with irradiation therapy, followed by surgical removal of the tumor if such is possible. Careful medical management of the myasthenia gravis during and after treatment of the tumor is mandatory.

SUMMARY

The relationship between thymomas and myasthenia gravis has been reviewed. Every patient with myasthenia gravis should have a roentgenographic examination of the chest, including lateral or oblique views for evidence of a thymoma.

Management of associated thymoma and myasthenia gravis is a difficult problem. The results have been best in patients treated with irradiation therapy followed by surgical removal of the thymoma.

Two cases with thymoma and myasthenia gravis have been seen at this hospital since 1947. One patient died of myasthenia gravis two years after irradiation therapy for an inoperable thymoma. In the other case, treatment of the thymoma by irradiation therapy, followed by surgical removal, resulted in almost complete remission of the myasthenia gravis.

REFERENCES

1. Eaton, L. M., and Clagett, O. T.: Symposium on myasthenia gravis; present status of thymectomy in treatment of myasthenia gravis. *Am. J. Med.* 19: 703-717, Nov. 1955.

2. Seybold, W. R.; McDonald, J. R.; Clagett, O. T.; and Good, C. A.: Tumors of thymus. *J. Thoracic Surg.* 20: 195-215, Aug. 1950.

3. Wilson, A., and Wilson, H.: Symposium on myasthenia gravis; thymus and myasthenia gravis. *Am. J. Med.* 19: 697-702, Nov. 1955.

4. Rowland, L. P.; Aranow, H., Jr.; and Hoefer, P. F.: Myasthenia gravis appearing after removal of thymoma. *Neurology* 7: 584-588, Aug. 1957.

5. Roe, B. B.: Myasthenia gravis secondary to thymic neoplasm; report of case in which symptoms developed 6 weeks after total thymectomy. *J. Thoracic Surg.* 33: 770-775, June 1957.

6. Madonick, M. J.; Rubin, M.; Levine, L. H.; and Karliner, W.: Myasthenia gravis developing 15 months after removal of thymoma. *Arch. Int. Med.* 99: 151-155, Jan. 1957.

7. Effler, D. B., and McCormack, L. J.: Thymic neoplasms. *J. Thoracic Surg.* 31: 60-82, Jan. 1956.

8. Grob, D.: Course and management of myasthenia gravis. *J. A. M. A.* 153: 529-532, Oct. 10, 1953.

9. Schwab, R. S., and Leland, C. C.: Sex and age in myasthenia gravis as critical factors in incidence and remission. *J. A. M. A.* 153: 1270-1273, Dec. 5, 1952.

10. Keynes, G.: Investigations into thymic disease and tumor formation (Cecil Joll lecture). *Brit. J. Surg.* 42: 449-462, Mar. 1955.

SERVICE ARTICLE

EFFECTS OF HIGH-INTENSITY NOISE ON NAVAL PERSONNEL

HALLOWELL DAVIS, M. D.

BEGINNING in 1952, several studies were made of the effects on naval personnel of the operation of jet airplanes from the decks of aircraft carriers, because questions had arisen as to what effect the exposure to high-intensity noise would have on the flight deck personnel. In 1953 a broad, exploratory study, now known as the BENOX study, was made of the biological effects of noise on man. The BENOX Report,[1] was reviewed and commented upon by the then newly formed Armed Forces-National Research Council Committee on Hearing and Bio-Acoustics (CHABA),[2] and recommendations were developed for research in this area. Both the BENOX Report and the CHABA commentary expressed the fear that exposure to jet engine noise in the unexplored range above 140 decibels might actually be producing injurious cumulative aftereffects. The Auditory and Non-Auditory Effects of High-Intensity Noise (ANEHIN) Project* was organized in April 1954 to study the problem that was stated so clearly in the above-mentioned reports.

NATURE OF THE PROGRAM

The project was conceived and planned as a field study, first to identify and thereby protect those individuals who might actually be suffering injurious effects, and second because it seemed impossible to bring into the laboratory both the noise exposures and the other stresses of actual military duty that

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*This work has performed by the Central Institute for the Deaf, St. Louis, Mo., with the collaboration of the U. S. Naval School of Aviation Medicine, Pensacola, Fla., under contract Nonr 1151 (02) with the Office of Naval Research. Central Institute personnel providing major services to the contract were: the author (Project Director), Dr. W. Dixon Ward (Experimental Psychologist), Dr. Donald G. Doehring (Experimental Psychologist), Dr. Robert W. Benson (Physicist and Electroacoustic Engineer), Dr. Jerome R. Cox (Physicist and Electroacoustic Engineer), and Mr. Arthur Niemoeller (Research Assistant).

might combine with noise to produce aftereffects such as chronic anxiety or undue fatigue. The location selected for the field study was the flight deck of aircraft carriers where the personnel forward of the island are exposed to particularly high noise levels. The study later was extended to include land-based maintenance personnel who serviced high-performance jet-powered aircraft. These two categories suffer the most severe sustained noise exposures regularly encountered in line of military duty.

Program Objectives

Except for the familiar impairment of hearing for high frequency sounds, the nature of the cumulative ill effects that were sought by the ANEHIN Project was not known in advance. The only clear specification was to look for undesirable *cumulative aftereffects*. It was never part of the ANEHIN objective to study the impairment of performance that may occur *during* noise exposure. This was conceived as a separate problem that might well be attacked as a laboratory experiment rather than as a field study. In other words, the ANEHIN Project was a wide-spreading "trouble hunt," in which the testing "net" was designed to cover as many aspects as possible. For this reason, a large variety of tests and procedures in addition to audiometry were employed to detect possible changes in sensory and psychomotor performance, equilibrium, central nervous and complex intellectual functions, anxiety, motivation, and the incidence of sick calls and accidents.

In planning the project, the inclusion of physiologic indicators of stress also was considered very carefully, particularly those that involve the activity of the adrenal gland, together with certain recognized medical indicators such as might be revealed by complete neurologic examinations. Another area of possible test and study was social and group behavior. Such studies were deferred, although not rejected outright, because of the elaborate and expensive nature of some of the procedures that would have been involved, on the one hand, and because, in the case of the physiologic indicators, it soon appeared that the degree of stress involved in the actual noise exposures was insufficient to create any real probability of obtaining positive results.

The ANEHIN Project actually became more than a search for the effects of high-intensity noise. It became a prototype of any similar trouble hunt that might be indicated at a later time in relation to any other new stress that may be created for personnel by future developments in the weapons and operations of modern warfare. The ANEHIN effort to carry out such a trouble hunt in the form of a field study has revealed certain difficulties and limitations inherent in any field study which, in spite of the consistently fine co-operation that the project received both on shipboard and on shore, seriously limited the scope and the significance of its actual findings. Field studies have their place, but they are expensive and time consuming and the un-

avoidable limitations imposed by operational considerations may seriously impair the scientific value of the entire effort.

ANEHIN CONTRIBUTIONS TO FUTURE RESEARCH

Test Batteries

As a positive contribution to any subsequent trouble hunt, the ANEHIN Project leaves a battery of physiologic, psychologic, and psychomotor tests and a group of questionnaires and pencil-and-paper tests of proved practicality and reliability. Significant progress was made toward the development of a standardized psychiatric interview and a rating scale based on the information so obtained. The tests cover a wide variety of functions and behavior. So wide is this variety that, in spite of the obvious holes in any system of screening, we believe that the probability of detecting undesired cumulative aftereffects of any sort is actually very high. Of course, in any other application, such as to the problems of vibration which may soon beset us in the future, special tests of any particular motor, sensory, or medical function that may be obviously endangered or already definitely implicated would be substituted for the audiometry that was emphasized in the ANEHIN Project.

New Test Equipment

As tools to be used in the search for cumulative aftereffects of high-intensity noise, a mobile laboratory and two new instruments were developed. All three may have a fairly wide application and significance in future work in this area.

1. *The mobile laboratory.* A mobile laboratory was developed to house the audiometric, the physiologic, and the psychologic apparatus, and to provide the proper environment for such tests in the field. In our trailer-mounted, sound-treated laboratory[3] (fig. 1), it was possible to make satisfactory audiometric measurements and to conduct psychophysiologic tests on the hangar deck of an aircraft carrier during training operations at sea. To provide adequate acoustic environment for audiometric testing in such a situation was no mean accomplishment from the point of view of accoustic engineering.

2. *The group audiometer.* One of the new instruments developed for this study is a group audiometer that employs the psychophysical method of single descent.[4] This instrument is designed to test 10 subjects at a time. It is semiautomatic, and in its full and final form includes an automatic print-out device that prints immediately the hearing losses of the 10 subjects in 10 typewritten columns (fig. 2). Audiograms for both ears of 10 men can be obtained in from 10 to 15 minutes. Fortunately, and somewhat unexpectedly, the method of single descent as incorporated in this audiometer has proved not only reliable but valid. The actual threshold values agree closely enough with the threshold values

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Figure 1. The floor plan of the mobile laboratory for auditory and nonauditory testing. The center room contains stools and testing desks for group tests of 10 men at a time. The laboratory has soundproofed walls and doors, and is air-conditioned throughout.

determined by the accepted standard methods of clinical audiometry to make it legitimate to consider the ANEHIN Group Audiometer for routine use in monitoring audiometry. Further

November 23, 1957 Class 46 57A Tester: J.W.G.

	1	2	3	4	5	6	7	8	9	10
250 R	30	00	42	42	30	42	36	00	36	46
	30	00	40	46	32	38	30	00	34	40
250 L	32	00	34	44	28	40	44	00	32	30
	32	00	36	42	28	38	40	00	34	36
500 R	48	00	56	54	44	54	62	00	46	56
	48	00	58	54	46	54	64	00	50	56
500 L	50	00	46	68	46	56	58	00	42	56
	48	00	48	66	40	54	56	00	50	56
1000 R	54	00	62	74	50	60	64	00	56	64
	54	00	70	76	44	62	68	00	58	66
1000 L	52	00	64	64	50	60	68	00	60	64
	52	00	66	78	42	60	68	00	56	62
2000 R	58	00	58	90	52	64	76	00	66	66
	58	00	58	72	56	58	70	00	62	64
	60	00	60	70	54	64	72	00	58	62
2000 L	60	00	64	76	56	50	70	00	60	62
	52	00	58	68	46	52	70	00	54	54
4000 R	40	00	62	78	44	54	70	00	58	60
	42	00	60	80	40	58	72	00	62	60
4000 L	36	00	58	68	42	54	58	00	66	56
	38	00	58	70	46	50	64	00	70	56
8000 R	00	00	26	54	00	50	42	00	46	48
	00	00	20	52	00	48	44	00	46	52
8000 L	16	00	18	52	14	44	48	00	48	50
	08	00	14	46	10	42	46	00	48	48

1 FMB
2 Unoccupied
3 DLW
4 JIC
5 JDH
6 LMR
7 WG
8 Unoccupied
9 WM
10 RWF

Figure 2. Sample data sheet from the Automatic Audiometric Data System (AADS). The two-digit numbers in the 10 columns are typed automatically. The operator makes all other entries. Readings are in thresholds expressed in decibels of attenuation below the starting level of the test tone. Reruns at each frequency demonstrate the repeatability of the procedure. Note that the occupant of Station 4 failed to push his "Don't Hear" button on the first trial of 2,000 cycles per second in the right ear, resulting in a reading of "90." The operator chose to repeat the test a third time at this frequency to verify the readings obtained on the second trial.

trial of the method with this end in view is being carried out at the U. S. Naval School of Aviation Medicine, Pensacola, Fla.

3. *The noise cumulator.* The second instrument developed for this study is called a noise cumulator. It is designed to measure the actual noise exposures incurred by personnel during military operations. It gives numerical measures of noise exposure in terms of the total duration of exposure to noise in specified frequency bands and above various preset intensity levels. The field use of the instrument involves tape recording the noise exposure experienced by a man during the actual performance of his job. This is accomplished by means of a frequency-modulated short-wave transmitter worn in the man's helmet, which broadcasts to the tape recorder those noises actually experienced at the wearer's head. Analysis of the tape recording is carried out later in the laboratory. The field unit, including microphone and transmitter, performs reliably and accurately in noise fields up to 150 db. The noise cumulator was used successfully on board the U. S. S. *Forrestal.* Considerable experimental work must still be done, however, to validate the measures obtained in terms of specified biological effects. Several different bases of acoustic analysis can be employed, depending on the choice of the method of rectification of the signal, the frequency bands, and the spacing of the intervals of intensity. Unfortunately, impulse type noises such as gunfire are still beyond the scope of the instrument. The particular measure or form of analysis must be found that correlates best with some specified effect. This will be a long and difficult task, but in the meantime the noise cumulator is already suitable for a basic survey of the actual current noise exposures that are associated with the operation of various aircraft and many other types of noisy equipment. If routine noise studies of the operation of new equipment are also carried out, any great increase in the noise exposure of service personnel can be detected or forecast in advance of actual field use.

THE ANEHIN FIELD STUDIES OF NAVAL PERSONNEL

During the years 1955 and 1956 many brief studies were conducted at the U. S. Naval School of Aviation Medicine to perfect the group audiometer and the various methods for psychologic, physiologic, and psychiatric testing. Considerable attention was devoted to the selection of tests suitable for use in the mobile laboratory under field conditions and to the collection of normative data. It was part of the original concept that a second laboratory at the U. S. Naval School of Aviation Medicine should also be maintained to develop new methods and to apply them to samples of naval personnel; and this type of work continues. Three field studies were successfully conducted.

The "Ticonderoga" Study

The first field study was carried out on board the U. S. S. *Ticonderoga* in two sessions, one during September-October 1955, and the other in May-June 1956. In 1955, preliminary sea trials were made using the mobile trailer laboratory. Audiometric measurements were made, and the various psychologic tests were tried out. In 1956, our psychiatrist colleagues, Capt. Philip B. Phillips, MC, USN, and Lt. William I. Stryker, MC, USNR, joined the ship in Istanbul, Turkey, at a time when the *Ticonderoga* had been operating in the Mediterranean for a period of 7 1/2 months. They administered questionnaires to and conducted standardized psychiatric interviews with a group of 70 selected personnel who had varying degrees of noise exposure in their different jobs. No definitive audiometric measurements or laboratory tests were conducted in connection with this second section of the study.

The "Forrestal" Study

The second study was carried out on board the U. S. S. *Forrestal* during its January-March 1956 training operations. In this study the feasibility of performing audiometry on board an aircraft carrier and also of measuring the noise exposure of personnel during operations by means of the noise cumulator was demonstrated. Psychologic and psychiatric tests were also successfully performed. This field study was more important as a demonstration of methods and their potentialities than as a source of definitive data, but the data and the qualitative impressions are all in agreement with those based on the two other studies.

The Cecil Field Study

Definitive studies of hearing and of psychologic performance were conducted in a third field study at U. S. Naval Air Station, Cecil Field, Fla., during the late summer of 1956 and early spring of 1957. The personnel selected for study in this case were chiefly maintenance personnel attached to various aircraft squadrons. The entire battery of tests, including audiometry, was employed in the summer of 1956 and then again after a period of approximately six months as a before-and-after study. The personnel included those who, by the nature of their jobs and the equipment with which they were associated, seemed to be the naval personnel incurring at this time the most severe noise exposures in terms of both intensity and duration. Control groups were selected from personnel who were not routinely exposed to jet noise.

LIMITATIONS OF THE ANEHIN STUDIES

Field Limitation

First, the ANEHIN study was a *field* study. The great advantage of a field study is that the results obtained are valid for the actual practical situation, but for this very reason it is more difficult to extract from the data any general statements or exact numbers. There are too many uncontrolled variables and extraneous factors, too much attrition of the original group in a test-retest study, and too little control of the major stimulus itself—in this case, the noise exposure.

Measurement of Noise Exposure

A second limitation is that there is no accepted, satisfactory measure of noise exposure. The noise exposures of flight deck and flight line maintenance personnel are brief exposures to noise of high but often rapidly changing intensity. The relations between intensity and duration for a given biological effect are unknown, and, until the noise cumulator is employed, the actual intensities and durations of operational exposures will not be known with any degree of precision. The independent variable, noise exposure, is therefore so vague that we cannot yet draw precise conclusions even when the dependent variable is measured numerically as decibels of threshold shift or as scores on well-standardized psychologic or physiologic tests. It was for just this reason, to define the noise exposures numerically, that the development of the noise cumulator was made a primary part of the project; but that development is only just now complete, and the present conclusions suffer accordingly in generality and in precision. We have been forced to rely on estimates of noise exposure based on questionnaires and on grouping men according to their jobs and according to the equipment used. We could do little more than give a rank order to the noise exposures of various groups as "high," "moderate," or "low," and sometimes only "more" or "less."

Neuropsychiatric Evaluation

Finally, in the area of neuropsychiatric evaluation the situation was even worse, because a neurologist's or psychiatrist's clinical judgment is a subjective opinion, based on many facts of observation and much previous experience. And it was to just this area of motivation, anxiety, fatigue, et cetera, that most of the anecdotal evidence of the BENOX Report pointed as the likeliest place where undesirable nonauditory cumulative aftereffects might be found. We therefore undertook to develop better methods of observation and evaluation, first by systematically standardizing the psychiatric interviews so that the same areas would be explored in all cases, and, second, by developing a psychiatric rating scale to make the evaluation of

the information more objective and reliable. This development is still incomplete, however, and, like the noise cumulator, it must still be validated. For the present, then, although the results obtained with the psychiatric rating scale are given in the full U. S. Naval School of Aviation Medicine Report, we still were forced to rely largely on the clinical impressions and judgments of our two psychiatrists. Fortunately, both of them were naval officers, and, therefore, had a background of appropriate clinical experience.

CONCLUSIONS AND RECOMMENDATIONS

Because of the limitations mentioned above, our results are neither all yes and no, nor sharply black or white. There are large gray zones of uncertainty in between, and because we cannot say "yes" it does not mean that the answer is necessarily "no." We do have at least partial answers to the following practical questions:

1. Should present operational procedures be modified, or are new types of protective equipment required, to protect military personnel from the cumulative ill effects of present noise exposures?

2. Are the new criteria for hazardous noise exposure too lenient? Are they too strict?

3. What of the future, with its expected increase in the noise levels of jet engines and other noise sources?

4. What recommendations emerge for the protection of personnel and for further study?

With practical questions, a precise scientific statement of fact does not constitute a complete answer. In addition, some kind of value judgment is involved or some extrapolation into the unknown is expected. The scientific data that we have, with our statistical analyses, will appear in a report to be issued by the U. S. Naval School of Aviation Medicine. The collective judgments of the ANEHIN group on the practical questions listed above, and a brief statement of the kind of data upon which each judgment is based, are as follows:

As of March 1957 there was no reasonable cause for immediate alarm concerning cumulative ill effects from the operational exposure of personnel to jet engine noise. We see no present need for changes in operational procedures or for protective equipment other than ear protectors of types that are currently available.

We have no evidence that the criteria of 1957 for hazardous noise exposures should be made more strict, nor can we say with assurance that they are too lenient. They lie within the zone of uncertainty of our conclusions.

Some of our findings suggest, but do not prove, that present operational exposures to jet engine noise may be near the limit of producing permanent impairment of hearing and also a general impairment of performance on a wide variety of psychologic tests. With increased noise exposure these effects may become not only clearly measurable but also of practical medical or operational significance. Efforts should therefore be continued to establish clearly the limits of tolerance for noise exposure by: (a) controlled laboratory experiments with volunteer subjects; (b) continued monitoring of actual operational noise exposures; (c) routine monitoring audiometry for all seriously noise-exposed personnel; (d) monitoring by selected psychologic tests of sample groups of the personnel who incur the most severe noise exposures; and (e) continual alertness of military medical officers to psychiatric or other medical indications of increased stress, either general or specific, associated with habitual noise exposure.

The most urgent research need is to develop a valid measure of noise exposure, such as the noise cumulator may provide, and to apply it to men during operational procedures with current and also with future noisy military equipment.

If adequate measures of actual and expected noise exposures are made, and *if* routine monitoring audiometry and also small sample psychologic and physiologic monitoring of highly noise-exposed personnel are instituted, then it will be legitimate to transfer some research effort from the problem of possible cumulative ill effects of noise exposure to the more urgent problems of the impairment of performance, including communication, in the actual presence of intense noise.

If and when a substantial increase in noise exposure occurs, due either to the introduction of noisier equipment or a change in operational procedure, a new study of cumulative effects should be undertaken by methods similar to those used in the present study.

ANALYSIS OF OBSERVATIONS

Noise Measurement

Measurements of the noise exposure experienced by several members of a flight deck crew were made aboard the U. S. S. *Forrestal* in March 1956. These preliminary measurements were concentrated on two members of the flight deck crew whose duties were such that they apparently were exposed to more intense noise than others. The measurements were accomplished by means of a miniature frequency modulation (FM) transmitter worn by the observer, an FM antenna on the flight deck next to the catwalk, and a fixed FM receiver and tape recorder located below deck. Figure 3 shows a record of the exposure of the forward plane director during a typical launch.

Cumulative distributions of noise level versus exposure time were obtained from a specially designed instrument, the noise cumulator. Operational exposures to very high-intensity noise

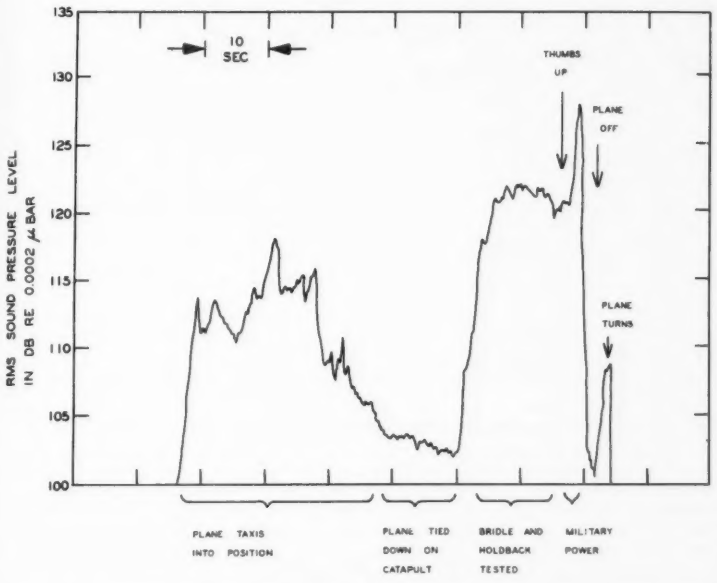

Figure 3. Over-all sound pressure level to which the forward plane director is exposed during a typical launch. The aircraft is the F7U-3 without operation of the afterburner.

are extremely brief (fig. 4). The duties of one of the men studied, the forward plane director, placed him in the center of the deck 20 to 50 feet behind the catapults. The second man studied was responsible for attaching the holdback mechanism to the rear of the airplane on the catapult. Both men appeared to spend more time than any others in the high-intensity regions of the sound field behind jets. However, neither man experienced peak levels in excess of 138 db for more than about 3 seconds during an average launch. As many as 100 launches in a day would produce less than 5 minutes' total exposure to such levels. Even exposure to peak levels above 115 db would total less than 1 1/2 hours daily.

This exposure to noise is significantly less than had been assumed in considerations of the possible hazards associated with noise exposure aboard aircraft carriers. The brevity and infrequency of exposure to extremely high noise levels has a very important effect on the noise exposure. This effect can be demonstrated by estimates of the equivalent steady exposure

experienced by a few members of the flight deck crew. The "equivalent exposure" is the level of steady noise sustained throughout an 8-hour day that would deliver the same total sound

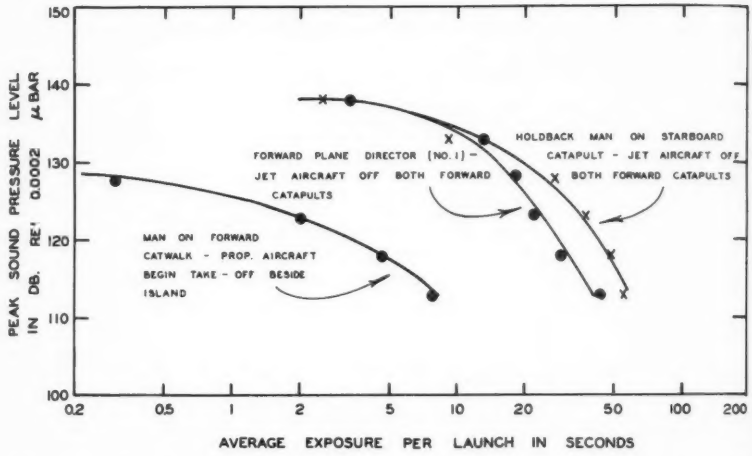

Figure 4. Cumulative distribution of peak-sound pressure level versus exposure time for two members of the flight deck crew. The exposure time is computed by averaging the results of several launchings. Data on exposure for a man stationed in the forward catwalk during the take-off of propeller aircraft is shown by way of contrast. These aircraft began the take-off roll near the island. The peak sound pressure level for jet engine noise is about 10 decibels (db) greater than the root mean square (rms) sound pressure level.

energy to the ear as does the actual noise exposure. An average daily launching of about 40 jet aircraft is assumed. The exact number launched is not critical since a change in this number by 50 per cent is no greater than the probable error of ± 1.5 db in the noise measurements. The equivalent exposure level is shown below for the two men studied in detail:*

	Equivalent rms SPL
Forward plane director - jet aircraft	105 db
Holdback man - jet aircraft	105 db

The estimates given above hold only for exposures to the particular combinations of planes aboard the U. S. S. *Forrestal* during March 1956. They assume that afterburners are not employed.

Auditory Effects

The most significant audiometric study in the ANEHIN Project was made at the U. S. Naval Air Station, Cecil Field, Fla. In

*The equivalent exposure level is in terms of the root mean square (rms) sound pressure level (SPL) re 0.0002 microbar. For typical jet engine noise the rms value is about 10 db less than the peak level.

August 1956 audiograms were obtained on 1,200 Navy enlisted men who were normal by otologic examination, including 579 men with some history of regular exposure to jet engine noise, and 379 with little or none. Among the latter were 91 men under 25 years of age who had not been exposed to large-caliber gunfire. These 91 men served as a "biological baseline." Their hearing was similar to that of similar groups in previous survey studies. In the initial audiograms, hearing losses were slightly greater among those men who were, or who had been, exposed to noise from reciprocating engines or from jets without afterburner. It is not certain from this cross-section study, however, that the engine noises had produced these hearing losses. Another probable cause of the greater hearing losses was gunfire. Almost twice as many of the "noise-exposed" personnel in the age groups 17 to 22 and 23 to 28 years had been subjected to large caliber gunfire. They also reported more tinnitus following their exposure to gunfire. The greater tinnitus suggested that, on the average, the severity of noise exposure may have been significantly greater for this group than for the "non-noise-exposed" group.

More significant than these moderate initial losses, however, are the follow-up audiograms taken of 220 of these same men in squadrons that were flying jet planes with afterburners. These were the men who presumably received the most severe noise exposures in the interval, yet they showed on the average *no additional hearing loss after seven months of routine activity.* A similar negative result also was found in our study of flight deck personnel during a three months' training cruise aboard the U. S. S. *Forrestal* in 1956.

The results of audiometric study at Cecil Field are presented and analyzed in considerable detail in an article by Ward.[5] Here the possible complications resulting from temporary threshold shift and from some increase in the use of protective equipment are considered carefully. A small additional impairment of hearing may have occurred during the seven months of noise exposure, but if so it was obscured by these factors that could not be controlled. The possibility that the men with initially greater hearing losses might be those who are more susceptible to incurring additional hearing loss from additional exposure also was examined, but no indication was found that men with pre-existing losses are more likely to suffer additional losses than men with normal hearing.

Obviously, the negative results of this study of men before and after seven months of routine operational exposure to jet engine noise is most reassuring. The results should not be interpreted, however, as contradicting in any way the criteria that have been established by other groups for hazardous noise exposure, particularly for exposure to steady industrial noises.

The criteria were developed on the basis of many years of noise exposure, while the present study extended for only seven months. The number of men who could be studied before and after this seven-month period under comparable conditions is not very large, and the expected slight trend toward increased hearing losses may be obscured by the many other factors that were involved. It is worth noting, however, that negative results very similar to the present ones have been obtained in other studies in which men exposed to aircraft noise have been monitored over a period of time. Sataloff,[6] in a five-year study of men working around jet engine test cells, found only slight changes, and even these could not be attributed unequivocally to noise. Similar negative results recently were reported by Kopra[7] in a study of Air Force personnel.

Nonauditory Effects

The search for nonauditory effects was also largely negative. In the preliminary studies aboard the U. S. S. *Ticonderoga* and the U. S. S. *Forrestal* no large differences were found between groups of noise-exposed and non-noise-exposed subjects. The final study at Cecil Field consisted of three parts: First, a group of 16 psychologic tests was administered twice to the same personnel, with an interval of six months between tests. Second, sick-call records were analyzed according to total frequency of reports to sick call. Third, a group of nine paper-and-pencil tests was given during the final testing period.

The battery of psychologic tests had been selected in order to sample as widely as possible a variety of different sensory, psychomotor, and intellectual functions. Twenty-five tests were employed in all: critical flicker frequency, tapping speed, reaction time, fine hand steadiness, gross hand steadiness, standing steadiness, and Knox Cube Test, fine dexterity, the Digit Symbol Test, tests of visual acuity, phoria, and depth perception, the Cornell Index, the Saslow Screening Inventory, the Taylor Anxiety Scale, and, in addition, nine factored aptitude tests that were given only at the end of the six-month period.

Analysis of the results of the initial tests given at Cecil Field in August 1956 showed no large differences between men having a history of exposure to jet noise and those having had no previous noise exposure. Our primary interest, however, was in assessing *changes* in test performance following the six-month period between tests as related to the severity of noise exposure during this period. A major difficulty in accomplishing this was the estimation of relative noise exposures of the different groups and individuals. Actually, three different estimates of noise exposure were employed. Subjects were divided into "most-exposed" and "least-exposed" groups on the basis of (1) the amount of noise generated by the plane which their squadron used, (2) the estimation (based on responses to a

questionnaire) of a man's noise exposure during the six-month period between tests, and (3) changes in his auditory threshold during the period between tests.

No positive relationships were found among the three above-named criteria for severity of noise exposure. That is, a man judged to be severely exposed by one criterion would not necessarily be judged to be severely exposed by either of the other two criteria.

In all, 95 of the men who were given the nonauditory tests at the beginning of the six-month period were retested at the end of the period. Six of the tests, which could be administered to 10 men at a time, were given to all the subjects. The remaining 10 tests, which required individual administration, were given to 31 of the men.

Each of the six tests that were given to all the men was analyzed according to each of the three exposure criteria, and in the resulting 18 statistical comparisons* there was only one case where the "most-exposed" group performed significantly worse (at the 5 per cent level of confidence) than the "least-exposed" group. The 10 tests given to the 31 men were analyzed only according to the criteria of individual exposure estimates and squadron assignment, because only four subjects in this group exhibited any hearing impairment. The 20 statistical comparisons that were made with this group of tests revealed no statistically significant differences between the "most-exposed" and the "least-exposed" groups.

The "most-exposed" group thus showed a significant deterioration of performance on only 1 out of 38 statistical comparisons. This one significant difference obviously could have occurred by chance, and it can safely be concluded that no adverse effects of noise exposure were demonstrated for this group of tests.

Although there is no statistical evidence of effects of exposure, nevertheless, on 26 of the 38 comparisons, the "most-exposed" group actually performed worse, even though only slightly worse, than the "least-exposed" group.

The reports to sick call by 183 men were analyzed for the total number of reports to sick call during the six-month period before the initial tests and also during the six-month period between tests. Changes in frequency of reports to sick call were analyzed according to the three criteria for noise exposure. The

*For the original analysis of nonauditory data the initial test score was subtracted from the retest score for a given test, and groups of subjects were compared in terms of absolute change in performance between the time of initial test and retest. Two of the discussants at the CHABA conference pointed out that use of the analysis of covariance technic would provide a more refined estimate of performance changes. Accordingly, all of the nonauditory data were reanalyzed by this method, and the results of the reanalysis are presented here.

"most-exposed" group showed a larger increase in frequency of sick call reports according to two of the three criteria, but none of the differences were statistically significant.

The results of the nine factored aptitude tests were analyzed according to an estimate of noise exposure from questionnaire information. Inasmuch as performance on seven of the nine tests should be correlated with intelligence level, the intelligence level of the groups was statistically equated by use of analysis of covariance. The "most-exposed" group gave the poorest performance on eight of the nine tests, but in only one case was the difference between groups statistically significant (at the 5 per cent level of confidence).

We can conclude with some confidence from the above analysis that the results in the nonauditory performances tested give no indication of a serious change for the worse as the result of greater noise exposure. Again the negative result is reassuring, even though the battery of nonauditory tests was not as extensive nor was the number of subjects tested as large as we had originally planned. The result is not entirely negative, however, because the "most-exposed" group performed worse, even though only slightly worse, than the "least-exposed" group on 36 out of a total of 50 comparisons.

Psychiatric Effects

In the psychiatric study aboard the U. S. S. *Ticonderoga* in May of 1956, the examinations and interviews were carried out when the *Ticonderoga* had been operating in the Mediterranean for 7 1/2 months. Due to unforeseen circumstances the cruise had twice been prolonged beyond the usual six months' time. During the 7 1/2 months, about 1,500 catapult shots had occurred.

A group of 70 men were selected, all with General Classification Test (GCT) scores above 30, who had all been present aboard the *Ticonderoga* for at least 75 per cent of the cruise. The majority of these men were flight deck personnel, although not all worked in close proximity to the forward catapults, and a small control group of men worked below decks with little or no exposure to noise. The men were classified with respect to noise exposure on the basis of their jobs. Thirteen had "very high" noise exposures, 18 had "high" noise exposures forward of the island structure, 13 had "moderate" noise exposures at or aft of the island, and 14 had relatively "low" noise exposures.

Psychiatric rating scores were obtained for each of these men by a rather elaborate method that cannot be described in detail in this article but which aims to divide the men into a "more adjusted" group and a "less adjusted" group. The mean scores of the four exposure groups differed but little, but when a cutting score was employed there seemed to be a significant

difference between the groups. The direction of the difference associated high noise exposure with deterioration of motivation or some increase in anxiety.

In addition to employing the standardized questionnaire and interview and the predetermined scoring system, the psychiatrists also noted the comments made by individual men and evaluated their reactions. Many of the subjects gave anecdotal reports similar to those in the BENOX Report. The most common complaints were increased irritability, tenseness, insomnia, and occasionally fear because of inability to communicate with other men in the presence of noise. With the exception of the difficulty of communication, however, most of the men stated that they did not believe that their trouble was due to the noise. They felt much more strongly that their trouble was due to the general dangers of the job and to further concern about a delay in their return to the United States that had been occasioned by a change in the schedule of operations of the ship.

The relative danger associated with the various jobs, therefore, was assessed. It appeared that there was indeed a greater degree of risk for the men in the very high noise exposure and the high noise exposure groups as compared with the low exposure group. The relation between the psychiatric rating scores and the probable danger associated with the job seemed to be at least as high as, and perhaps higher than, the relation between rating scores and noise exposure. This conclusion, reached by the psychiatrists from their own observations, was borne out by the statements made by the subjects themselves. Among the men working in the most dangerous jobs (involving also very high noise exposures) 92.3 per cent expressed anxiety about their jobs, while among the men working on the safest jobs just below the flight deck only 21.4 per cent expressed any anxiety about their jobs. Very few men, however, even among the high noise-exposure group, expressed the opinion that the jet noise was disturbing to them.

The over-all subjective impression developed in May 1956 by the two psychiatrists aboard the U. S. S. *Ticonderoga* was that the anxiety level among the men working on the flight deck was considerably greater than what they had encountered among a similar group of men interviewed aboard the same ship in the previous September, about eight months earlier. Unfortunately the overlap between the earlier group studied and the final group was too small and the differences and the methods of interview employed were too large to allow any significant comparison of scores for the same individuals before and after the 7 1/2-month cruise.

It is the over-all opinion of the psychiatrists that the basic reasons for the increased anxiety revealed in the psychiatric

interviews lay in the greater awareness of the inherent dangers of the jobs and in the fact that the ship was overdue for return to the States. The awareness of danger had undoubtedly been heightened by several fatal accidents that had occurred on the flight deck during the cruise. Furthermore, whatever the neuropsychiatric effects of high noise exposure may be, they are subtle and difficult to extract. The present series of carefully planned and carefully conducted interviews did not reveal any easy or obvious correlations with noise exposure, nor do they give any cause for alarm.

The general conclusion from our auditory, nonauditory and psychiatric observations is, therefore, that no one of the many functions examined in this series of nonauditory tests stands out clearly as being adversely affected by noise exposure according to any one of the three admittedly imperfect criteria used for estimating the severity of the noise exposure. There is, however, a definitely suggestive trend in the direction of a slight over-all decrement of performance associated with the most severe noise exposures. Noise levels as of March 1957 did not constitute a serious threat for exposed personnel in terms of large changes in performances on our test battery. Because of the suggested trend toward a slight general decrement, however, we must view with some concern the future effects of higher noise levels and longer periods of exposure. The negative conclusions apply only to a period of six months' exposure to noise sources as powerful as the F4D.

FINAL EVALUATION

The over-all result of the ANEHIN survey is definitely reassuring. Some initial auditory impairment was found among naval personnel, as in the previous survey, but it could not be proved that six months' operational exposure to jet engine noise, even with afterburners, increased the auditory impairment. None of the nonauditory tests showed any really clear correlation between worse performance and greater exposure to jet engine noise. There was a slight increase in the number of sick calls and a trend toward a very general nonspecific lowering of test performance, but these were merely trends. An increase in anxiety and tension was found among flight deck personnel near the end of a 7 1/2-month Mediterranean cruise, but the anxiety and tensions were probably caused by hazards and stresses other than jet engine noise.

Why, after the solemn warnings of the BENOX and CHABA reports, was so little evidence of any cumulative injurious effects of exposure to the high-intensity noise of jet engines found?

There are three definite and one rather vague answers to this question. (1) The increase in the noise produced by operational

aircraft in the fleet did not occur as soon as appeared likely when the BENOX and CHABA reports were written. One particularly noisy type of airplane never became operational and another was slow in reaching full employment. (2) The introduction of more powerful catapults had greatly reduced the use of the afterburner at take-off. This has reduced the anticipated noise exposure on the carrier deck by several decibels. (3) The really severe noise exposures on the flight deck were, and still are, very, very brief.

It is for these three reasons that the ANEHIN Project found itself in the field in good time, ahead of the great anticipated increase in noise exposures on shipboard. For these reasons ANEHIN could properly transfer its activities ashore to the U. S. Naval Air Station, Cecil Field, Fla., where the maintenance men who service jet planes with afterburners actually incur much more severe and prolonged noise exposures than do the carrier flight deck personnel.

Even among the shore-based maintenance men, the results are almost, although not quite, negative. This is sheer good fortune. The vague conclusion is that Nature's margin of safety proves to be a little wider than the authors of the BENOX report feared it might be. For the present, then, reasonable caution, observance of criteria for noise exposure, use of personal protective equipment, and the institution of appropriate monitoring procedures should suffice. How wide the present margin of safety may be we do not know, but for the present we believe that further field studies of cumulative aftereffects are not needed except as part of a sampling, spot-check type of monitoring. If the limits of tolerance for high-intensity noise exposure are to be sought, the job can be done much more cheaply and effectively under controlled conditions, as in a laboratory, than as a field study.

Our final words are again a reassurance as to the present situation, followed by a warning against overconfidence and complacence in regard to the future. We still have not encountered the noise stresses foreseen in the first CHABA Report.

DISCUSSION

Doctors Nello Pace, R. C. Davis, Lyle Jones, David McK. Rioch, and William Burns briefly discussed the report.*

Doctor Pace remarked that relatively simple tests of adrenal cortical function based on blood and urine samples might detect underlying

*Dr. Pace is at the Department of Physiology at the University of California, at Berkeley; Dr. R. C. Davis is in the Department of Psychology at Indiana University; Dr. Lyle Jones is in the Psychometric Laboratory at the University of North Carolina; Dr. David McK. Rioch is the Director, Neuropsychiatry Division, Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington, D. C.; and Dr. William Burns is at the Charing Cross Hospital, London, England.

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physiologic changes, even though the person appears to be unaffected in terms of his ability to perform under stress. Doctor R. C. Davis noted that the autonomic nervous system may participate in the adjustment of the body to stress and the maintenance of performance. Doctor Jones commented that the statistical technic of analysis of covariance would partial out undesired variables, especially those associated with the unreliability of a given physiologic or psychologic measure. He suggested that the use of this technic in recalculating some of the statistical data would result in much more meaningful and useful data. Doctor Rioch added that field studies often have as their major result the identification of the relevant factors in the problem and the observations that need to be made. In addition, new apparatus is frequently developed that has applications beyond the situation for which it was created. The ANEHIN noise cumulator is a major contribution to the study of noise exposure in general.

Doctor Burns briefly reviewed the British Navy's research on hearing conservation in high-intensity noise. The Royal Navy is placing great stress on educating service personnel on the necessity of protecting their hearing in the presence of high-intensity noise. Otherwise, the British work is of the same general character as the American effort, with much the same conclusions. The American V-51R ear plug is now being superseded on the carrier flight deck by the British MK-1 helmet equipped with an ear muff that incorporates a leather sealing ring as well as an acoustic valve. Other improved models of the muff are now being introduced.

SUMMARY

Carrier flight deck personnel on board the U. S. S. *Forrestal* and the U. S. S. *Ticonderoga,* and shore-based jet aircraft personnel at the U. S. Naval Air Station, Cecil Field, Fla., were tested by pure-tone audiometry, psychologic and psychomotor performance tests, group paper-and-pencil tests, psychiatric interviews, and analysis of sick-bay calls. The results were related to the estimated relative noise exposures of the various exposed and control groups tested.

The audiometry was conducted in a specially designed and constructed trailer laboratory by means of a group audiometer that was developed as part of the project. This semiautomatic instrument is suitable for monitoring audiometry elsewhere.

A method for the measurement of the total noise exposure of individuals during actual military operations or maintenance tasks was developed. This method relies upon a special device, the "noise cumulator," which was designed, constructed, and a prototype tested on board a carrier. The complete device was not, however, available until after the three field studies. This instrument is suitable for monitoring the noise exposures to be expected from future aircraft.

The battery of psychologic and psychomotor tests was composed of simple, well-tried tests of known significance that sampled a wide variety of sensory, motor, intellectual, and emotional functions.

No clear, positive effects, either auditory or nonauditory, of exposure to noise were shown by any of our tests. Certain small positive effects were found by the audiometric and the psychiatric studies, but these are attributed to causes other than jet engine noise. A suggestive trend toward a slight general decrement in performance did appear in the psychologic tests of jet plane maintenance personnel.

CONCLUSIONS

As of March 1957, there was no reasonable cause for immediate alarm concerning cumulative aftereffects of jet-engine noise exposures of naval personnel.

It is unsafe to extrapolate from present noise exposures to the more severe exposures that must be anticipated in the future. The extent of the present margin of safety is not known, and continued vigilance is required.

In the opinion of the ANEHIN group, at least two other undesired effects of high-intensity noise were, as of 1957, more serious threats to military operations than were its cumulative aftereffects. They were (1) interference with communications, and (2) decrement in performance of personnel during actual exposure to high-intensity noise.

RECOMMENDATIONS

Further field studies of this sort are not necessary until there is a considerable increase in operational noise exposures. Meanwhile, monitoring of noise exposures with the equipment developed by ANEHIN should be instituted to detect such changes.

Monitoring audiometry and also monitoring by selected psychologic and psychomotor tests should be instituted for the protection of personnel who suffer the most severe noise exposures.

Valid criteria of hazardous noise exposures should be established by carefully controlled experiments with human volunteers, using careful measurements of noise exposure and the tests of the ANEHIN battery. In such experiments the decrement of performance during actual exposure to noise also should be measured.

REFERENCES

1. *BENOX Report*, an Exploratory Study of the Biological Effects of Noise. Contract N6 ori-020 Task Order 44, ONR Project NR 144079, The University of Chicago, Ill., 1953.

2. *CHABA Report No. 1*, High-Intensity Noise and Military Operations: an Evaluation, Technical Report No. 1 from the Central Institute for the Deaf to the Office of Naval Research under Contract Nonr 1151 (01), NR 140-069.

3. Cox, J. R.; Benson, R. W.; and Niemoeller, A. F.: Mobile laboratory for group hearing tests. *Noise Control* 3: 44-48, 92, Mar. 1957.

4. Ward, W. D.: Single-descent group audiometer. *Noise Control* 3: 15-18, May 1957.

5. Ward., W. D.: Hearing of naval aircraft maintenance personnel. *J. Acoustical Soc. Am.* 29: 1289-1301, Dec. 1957.

6. Sataloff, J.: Effect of prolonged exposure to intense noise on hearing acuity. *A. M. A. Arch. Otolaryng.* 58: 62-80, July 1953.

7. Kopta, L. L.: Hearing loss among Air Force flight-line personnel. *J. Acoustical Soc. Am.* 29: 1277-1283, Dec. 1957.

SPINNING IN A SPACESHIP

"From observation of the V-2 and Viking it appears that these rockets spin fairly rapidly and unpredictably on their upward flight and simple analogy shows what the effect of this might be on the human body. If you spin round and round while standing in the one position and then stop, what happens? Everything else in the room appears to spin and you probably feel giddy. If you now try to pick something off the floor or table you experience great difficulty in doing so. This is what would happen in the rockets of today only to a greater degree. If the spinning occurs at right-angles to this, that is if the crew were seated in the spaceship, then spinning would be round an axis fore and aft through the body at the lower end of the chest. In this situation consciousness is lost fairly quickly and death might even occur. Although spinning is obviously a serious hazard it is one which can be avoided by design, and in the future rockets will require to be stabilized."

—M. KENNEDY BROWNE, B. Sc., M. B., CH. B.
in *Spaceflight*
p. 142, July 1957

Clinicopathologic Conference

Tripler U. S. Army Hospital, APO 438, San Francisco, Calif.*

DYSPNEA AND DYSPHAGIA IN INFANCY

Summary of Clinical History. A two-month-old Caucasian-Oriental male infant was admitted to this hospital from the Pediatric Clinic on 17 July 1954. He previously had been hospitalized on 20 May, when 11 days old, for severe diarrhea. At that time his weight was 4 pounds 8 ounces, a decrease from his birth weight of 6 pounds 3 ounces. At the time of his discharge on 4 June, he weighed 6 pounds. He was seen regularly in the Pediatric Clinic. His mother stated that he had been doing fairly well at home, but within the past two weeks she noted a decreased fluid intake from approximately 30 to 35 ounces per day to around 20 to 22 ounces per day. There was moderate dyspnea associated with feeding. He was last seen in the Well Baby Clinic one week prior to admission to this hospital, when his weight was 8 pounds 8 ounces. The mother said that during the three or four days immediately prior to admission, his fluid intake had further decreased to the point where he was taking from 1 to 3 ounces per feeding. When seen in the Pediatric Clinic the morning of admission, he was in poor physical condition. He weighed 8 pounds.

Physical Examination. On admission the infant's temperature was 100.4°F. Physical examination revealed a moderately dyspneic, small male infant who appeared "somewhat grayish and pallid." There was no evidence of dehydration. He seemed fairly alert and in good contact with his environment. The only significant physical findings recorded at the time of admission were scattered moist rales over the left apex, anteriorly and

*At the time of this conference, Maj. Gen. John F. Bohlender, MC, USA, was Commanding General. From the Pathology Service, Col. Harold E. Shuey, MC, USA, Chief.

posteriorly, and a cardiac rate of 148 per minute without murmur or thrill. The liver and spleen were not palpable. Remaining physical examination, including neurologic tests, was within normal limits.

Laboratory Studies. A blood cell count on 17 July showed a hemoglobin of 10.0 grams per 100 ml; erythrocytes, 2,750,000 per μl; white blood cells, 16,700 per μl with 48 per cent neutrophils, 51 per cent lymphocytes and 1 per cent monocytes. A repeat blood cell count on 21 July was reported as showing hemoglobin, 10.4 grams per 100 ml; red blood cells, 3,240,000 per μl; white blood cells, 28,000 per μl with 39 per cent neutrophils, 51 per cent lymphocytes, 7 per cent monocytes, and 3 per cent eosinophils. Urinalysis revealed a trace of albumin, rare red blood cells and 4 to 8 white blood cells per high power field. Blood chemistry study on 17 July revealed carbon dioxide combining power of 20.2 mEq per liter.

The report of the roentgenographic examination of the chest on 17 July read, "No infiltration is recognized." A repeat examination the next day was interpreted, "No infiltration is recognized; the stomach is greatly distended with gas." Another chest film on 23 July showed mottled shadows over the right upper lobe and right lower lobe with slight deviation of the trachea to the right. The changes were considered consistent with an aspiration pneumonia and some atelectasis in the right upper lobe. An examination was made on 27 July with introduction of contrast medium into the esophagus. The report was, "The esophagus was normal in course and caliber; no vascular ring or obstructing defect in the mediastinum was observed." Six repeated stool examinations for ova, parasites, and enteric pathogens were reported as negative.

Course in Hospital. Because of the inconstant rales over the left apex, administration of 279 mg (300,000 units) of procaine penicillin G daily was begun. Two days after admission, the infant had an episode of gagging and coughing associated with a feeding and became cyanotic for about 25 minutes. Following this, the penicillin dosage was increased to 198 mg (200,000 units) every 6 hours, and 50 mg of streptomycin sulfate every 6 hours was added. The temperature remained elevated to 100° F for the first 48 hours, after which it subsided and remained normal throughout the remaining hospitalization.

Because of the infant's generally poor condition and the hemoglobin of 10 grams, he was given a transfusion of 50 ml of whole blood on 21 July, and 40 ml on 22 July. His clinical appearance improved somewhat following the transfusions, but he continually demonstrated a trace of cyanosis.

On 23 July, because of the continued respiratory distress and dyspnea, the patient was seen in consultation by the Otolaryngology Service. The consultant believed that because

of the child's poor condition, endoscopic examination was not advisable at that time. The infant was treated conservatively for the next three days, but the apparent respiratory obstruction at about the level of the larynx continued. The nurses noted that the child took his feedings best when lying on his abdomen with his head turned to one side or the other. Feedings every three hours were instituted on 26 July. The baby began to eat more avidly and to take larger amounts, averaging approximately 150 ml at each feeding.

On the morning of 27 July when the child appeared no better, he was taken to the x-ray department where Lipiodol (brand of iodized poppy seed oil) studies of the esophagus were done. Injection of the contrast medium was made through a No. 6 French catheter, placed with considerable difficulty into the upper portion of the esophagus. The report was as stated above. Later that same afternoon it was decided to attempt direct laryngoscopy. At approximately 1530 hours, the child was taken to the Ear, Nose, and Throat Clinic. The laryngoscope was introduced and when it reached the base of the tongue, respirations ceased. The infant was returned to the ward and artificial respiration, continuous tracheal aspiration, administration of oxygen by mask, and intracardiac injection of caffeine sodium benzoate were all employed without benefit. The cardiac action ceased at approximately 1605 hours.

DISCUSSION

Doctor Bruton:* This is the story of a two-month-old infant who was first hospitalized at 11 days of age because of severe diarrhea of unknown cause. We are told that he lost 27 per cent of his body weight, or what would amount to approximately 40 pounds for an average-sized adult, because of this illness. In spite of his severe disease he was doing well enough to be discharged after two weeks. He was thought to be fairly well at home for two to three weeks and then it was noted he was feeding poorly and having moderate dyspnea with each feeding. The latter symptoms increased in severity over a two-week period so that on the second admission he was obviously in poor condition, gray and pallid, with moist rales scattered over the left upper lung field. His temperature was only slightly increased on admission and his entire 10-day hospital course was characterized by the lack of fever. His white blood cell count was moderately increased to 16,700 and 28,000 per μl with only 48 per cent and 39 per cent neutrophils. The roentgenogram is reported to us as showing no infiltration at that time. We will look at it later, as we should on all our patients.

However, let us pause here to consider the possible causes of the symptomatology as given. Certainly, the most common, and a very common one, is a respiratory infection, often resulting in the young infant in obstructive emphysema, in which there is a marked bronchial

*Col. Ogden C. Bruton, MC, USA, Chief, Pediatric Service.

reaction, perhaps with areas of pneumonitis and atelectasis. We call this acute bronchiolitis; others may call it acute interstitial pneumonia, and in its milder form and in older infants and children, it may be called asthmatic bronchitis. These conditions are usually much more acute than described in this infant and may be so fulminating as to result in death in a few hours. Although upper respiratory infection may precede lower respiratory symptoms, such an event rarely occurs in such an insidious manner as in this infant. Rales, fine and diffuse, are almost regularly heard, and roentgenograms usually show depression of the diaphragm with other evidence of emphysema which may indeed cover up any other process present in the lungs. Although this infant may have had a mild respiratory infection, we do not have the usual findings associated with this type of fatal respiratory disease.

With cyanosis and dyspnea we must mention congenital heart disease; but in the absence of murmurs, change in heart size, and lack of polycythemia we really have no clues to lead us to a serious consideration of a cardiac lesion.

A bilaterally obstructed nose may lead to marked distress in young infants. This may occur during the first hour of life if congenital, or later if due to swollen mucous membranes from infection or other insults. It is not unusual for a week-old infant to be admitted to a pediatric ward in marked distress, thought to have cyanotic heart disease, and by simply opening his mouth, have all the signs disappear. The observers of this infant did not believe that obstruction was present in the nose, and we can accept this to rule it out.

From the two extremes already mentioned, our attention is focused on a more middle ground around the larynx inasmuch as those observing the patient seemed to think he had a laryngeal obstruction. In this age group one naturally thinks again of congenital defect. The clinicians properly excluded a vascular anomaly constricting the trachea and esophagus by observing a contrast medium take a normal undisturbed course down the esophagus. Other congenital defects like stenosis of the larynx and trachea and the usual forms of tracheoesophageal fistula with esophageal atresia would be evident shortly after birth. We have to postulate then, an acquired lesion and/or a lesion which gradually encroached more and more on the infant's airway and esophagus. Various tumors of the larynx could do this: hemangioma, adenoma, papilloma, cystoma, fibroma, lipoma, lymphangioma, chondroma, et cetera. These must be extremely rare in infants of this age, and lacking any positive evidence for their diagnosis, the more common type of "tumor" caused by infection must be given first consideration. The course of retropharyngeal abscess appears to be consistent in most details given us with the course of illness observed in this infant. It is distinctly a disease of infancy, most cases occurring in the first year of life, and it is almost never seen after the age of three years. Origin is usually a sequel to acute infection in the nose and throat. Infection localizes in the retropharyngeal lymph glands which overlie the second and third cervical

vertebrae. Increasing enlargement of these glands and surrounding area leads to increasing dyspnea and dysphagia. The course may be rapid and the abscess rupture into the pharynx in two to three days, or the pus may dissect downward along facial planes to the posterior mediastinum or laterally into the deep tissues of the neck. Inasmuch as a unilateral cervical gland enlargement is almost regularly found and is of considerable diagnostic value, it is surprising no mention is made of such a gland in this case. These infants are usually very toxic, have considerable fever, and appear to breathe better by retracting the head, a finding which may suggest meningitis to the examiner—one wonders if such a position was being described by the nurses' notes mentioned earlier. The swelling may be seen by inspection and felt by an educated finger. Once the diagnosis is suspected it may be confirmed by a lateral roentgenogram of the neck, including the pharynx and chest.

May we see the roentgenograms on this infant?

Doctor Harrell:* The films made on 22 May at the time of the first admission, show an extensive right upper lobe pneumonia which is visible only on the posteroanterior film (fig. 1). On the lateral view, the infiltration is hidden under the scapula and cannot be seen. In the next films, taken on 17 July at the time of the second admission, there is no pulmonary infiltration, but one can see a retropharyngeal mass which is quite large, actually the size of a small orange or a lemon (fig. 2). There is bony destruction of at least two cervical vertebrae that suggests an osteomyelitis. So the question is raised as to whether this whole process could be secondary to an osteomyelitis that could have followed the pneumonia of two months earlier. Films taken on 23 and 24 July are of no assistance because the neck is flexed, causing the trachea to buckle. One can see on these spot films that the tube used to introduce contrast medium into the esophagus has been deviated to the left side of the trachea. This is a significant finding, but because of the density of the films it takes examination with very bright light to tell this.

Doctor Delaney:** It appears to me from back here that the pedicles of the vertebrae are widened. Can we be sure that this is not an anterior meningocele?

Doctor Bruton: Dr. Harrell, can you tell us whether or not the involvement of the spine is an anomaly or is it a destructive process, probably due to infection? If this represents an anomaly of the spine, the tumor would more likely represent a cyst, possibly filled with spinal fluid or, in other words, an anterior spina bifida cystica—a most unusual lesion to occur at this level of the spine. Other types of cystomas arising from remains of secreting tissue could and are more likely to make their appearance in this area.

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*Col. Henry C. Harrell, MC, USA, Chief, Radiology Service.
**Lt. Comdr. Thomas B. Delaney, MC, USN, Pediatric Service.

Doctor Harrell: I do not think that the pedicles are particularly widened, and there is no indication on these films of any congenital anomaly. Besides, the first films clearly show that the cervical vertebrae are normal. It is only in the later films that destruction of these vertebrae is seen.

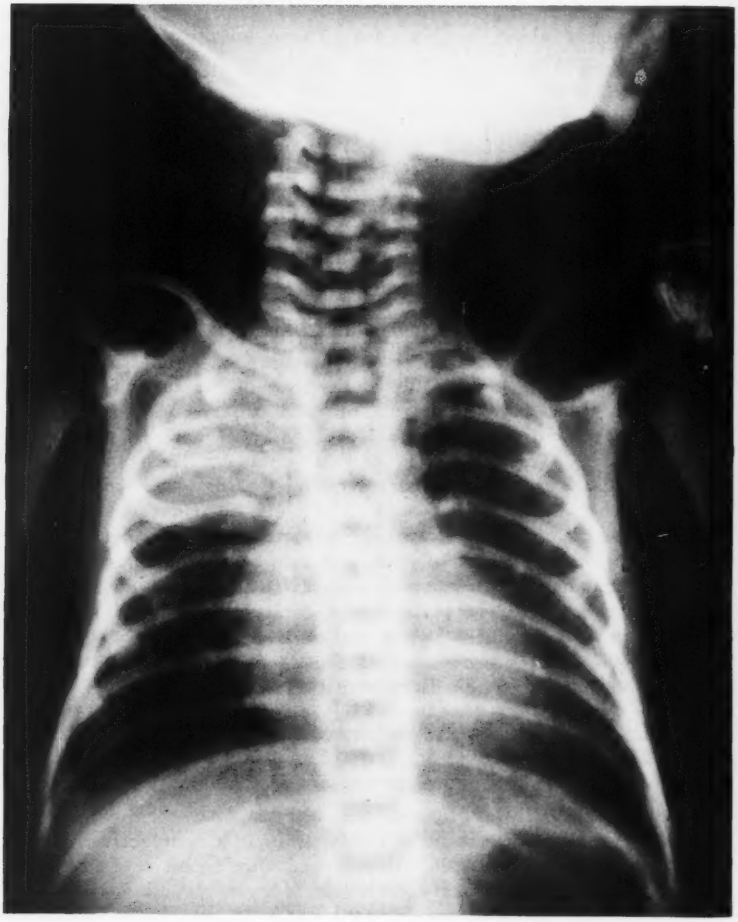

Figure 1. Roentgenogram of chest on 22 May 1954, showing right upper lobe pneumonia.

Doctor Bruton: Thank you, Dr. Harrell. That is great help to us. The afebrile course, the lack of toxic signs, the slow progression, the low neutrophil count, and absence of a one-sided cervical lymph gland enlargement would all fit well with an aseptic cystic process. However, since we can be quite sure from what we have learned from

the roentgenograms that the process in the spine is an infectious one, we can feel fairly certain, in spite of what we have said against it, that the tumor represents an abscess. Symptomatically, from the obstructive standpoint, the two may be similar.

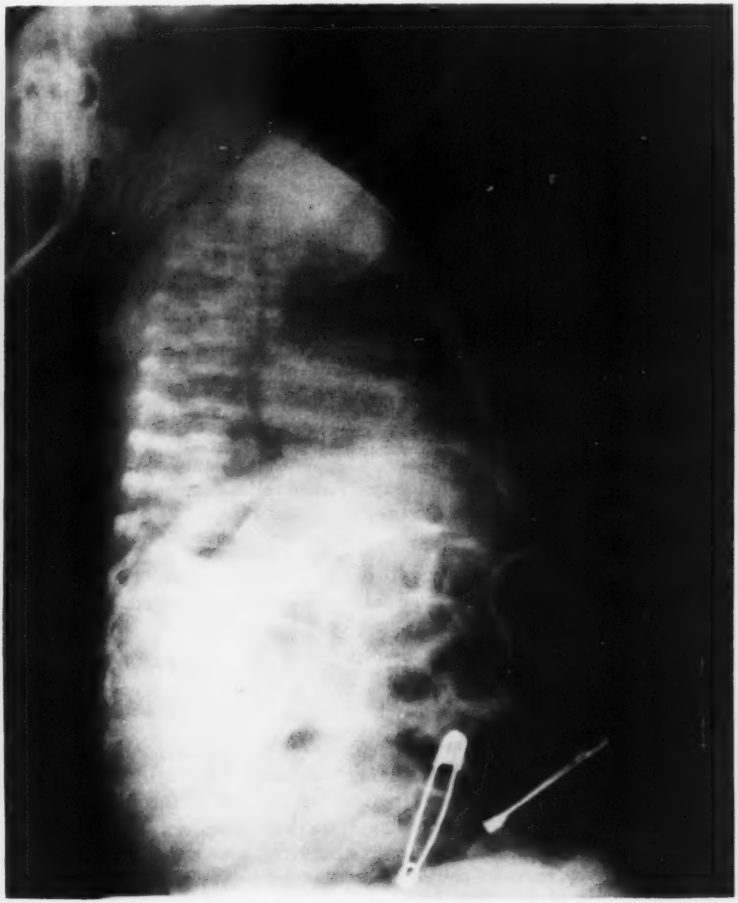

Figure 2. Roentgenogram of infant on 17 July 1954, showing anterior displacement of trachea by bulging posterior pharyngeal wall, and destruction of cervical vertebrae.

Doctor Harrell: Yes, but was he not getting penicillin all along?

Doctor Bruton: We are only told that the patient had penicillin after his second hospital admission. He may have had more fever before this time. I am considerably bothered by the fact that this child had no cervical lymph nodes, a finding which is almost constant in retropharyngeal abscess.

It is not unusual for death to occur suddenly from rupture of the abscess and flooding of the lungs. Asphyxia may develop by closure of the airway if swelling overlies or involves the larynx. The third way death may occur is from a so-called "vasovagal reaction" producing cardiac standstill at the time of examination, as appears to have occurred in this infant. That the process did indeed rupture in this infant is suggested by the statement that he was given "continuous tracheal aspiration."

A chronic type of retropharyngeal abscess occurs with tuberculosis of the cervical spine. This infant is quite young to have tuberculosis; however, inasmuch as his abscess developed rather slowly, without much fever and with evidence of cervical vertebral involvement, one must consider the possibility. We have no report of a tuberculin test or history of exposure to tuberculosis and therefore cannot reach this conclusive diagnosis.

It also is tempting to connect his diarrheal disease at 11 days of age with the fatal abscess. Any organism could be the agent, with past statistics showing streptococcus found most frequently. It is suggested, however, that his diarrheal disease may have been the result of a Salmonella infection, which sometimes invades bone, and that his abscess which subsequently developed was caused by that organism and may have been found in the abscess at autopsy.

Doctor Marcarelli:* When you say "Salmonella diarrhea" in this age group, do you include typhoid?

Doctor Bruton: Either typhoid or paratyphoid organisms can localize in bone and cause an osteomyelitic abscess; however, I had in mind the paratyphoid which is frequently isolated in diarrheal disease in this area.

Doctor Harrell: I think your concept that he acquired both of the diseases at the same time is very sound, but from the roentgenographic point of view, this could possibly be tuberculosis of bone.

Doctor Bruton: We have chest films on this patient for some period of time, and at no time after 22 May was there any indication of pulmonary tuberculosis either in a peripheral lung or in the hilus. I think this speaks against tuberculosis. I do not see how this could be called a tuberculous infiltration in the first film at only 11 days of age and, if so, how it could have cleared so completely in subsequent roentgenographic examinations.

Doctor Harrell: I would agree; I do not think it is tuberculosis either.

Doctor Bruton: In summary, we have a two-month-old infant who had a severe diarrheal disease and pneumonia at 11 days of age from which he appeared to recover only to develop increasing dyspnea and dysphagia after two weeks at home. Lateral roentgenograms of the pharynx revealed a "tumor" in the posterior pharyngeal area, as well as destructive changes in the upper cervical vertebrae. The infant died

*Capt. Joseph L. Marcarelli, MC, USA, Resident, Department of Medicine.

suddenly during examination, which is common in infants with retropharyngeal abscess, and which this infant appears to have had. He died either from drowning in pus, from complete closure of the airway, or vasovagal cardiac standstill.

Clinical diagnoses:

1. Retropharyngeal abscess
2. Laryngeal edema

Dr. Bruton's diagnosis:

Retropharyngeal abscess with possible rupture

PATHOLOGIC FINDINGS

Doctor Shuey: At autopsy, the body was that of a well-developed, well-nourished Mongolian male infant about two months of age. The oral mucous membranes exhibited a grayish pallor. There were bilaterally enlarged, firm, cervical lymph nodes, a finding not recorded in the physical examination. A firm, bulging mass was palpable in the posterior pharynx, which was found to occupy the retropharyngeal space down to about the level of the first thoracic vertebra. At this point, the trachea and esophagus were displaced anteriorly and to the right. The mass was produced by an abscess cavity which contained about 50 ml of thick, yellow-green pus from which abundant coagulase-positive *Staphylococcus aureus (Micrococcus pyogenes* var. *aureus)* was cultured.

The only other gross findings of interest were focal atelectasis and emphysema of the lower lobe of the left lung, congested liver and kidneys, and a Meckel's diverticulum.

Microscopically, a section through the trachea, esophagus, and lateral lobes of the thyroid revealed the abscess wall to consist of edematous granulation tissue infiltrated with neutrophils, lymphocytes, plasma cells, and histiocytes (fig. 3). The inflammation involved the supporting tissues but did not infiltrate the esophagus, trachea, or thyroid gland (fig. 4).

A section of the lower lobe of the left lung substantiated the gross impression of atelectasis and emphysema (fig. 5).

Death was attributed to respiratory obstruction during attempted passage of a laryngoscope. The abscess had not ruptured and no pus was present in the tracheobronchial tree. A vasovagal reaction could not, of course, be demonstrated anatomically, had such an event occurred. The exact mechanism of death is still obscure. Laryngeal edema was not apparent. The atelectasis in the lower lobe of the left lung was not, in itself, sufficient to cause death. The posterior pharyngeal wall, in addition to bulging toward the larynx, was markedly edematous, as were the supporting structures of the neck organs. This could have been aggravated by the instrumentation to the point of causing virtually complete respiratory obstruction. The congestion of the liver and kidneys probably was an agonal event. The Meckel's diverticulum was an incidental finding.

Jackson and Jackson,[1] in "Diseases of the Nose, Throat, and Ear," admonished the reader to exercise care in the treatment of these cases, as incision of the abscess may result in flooding of the pharynx with

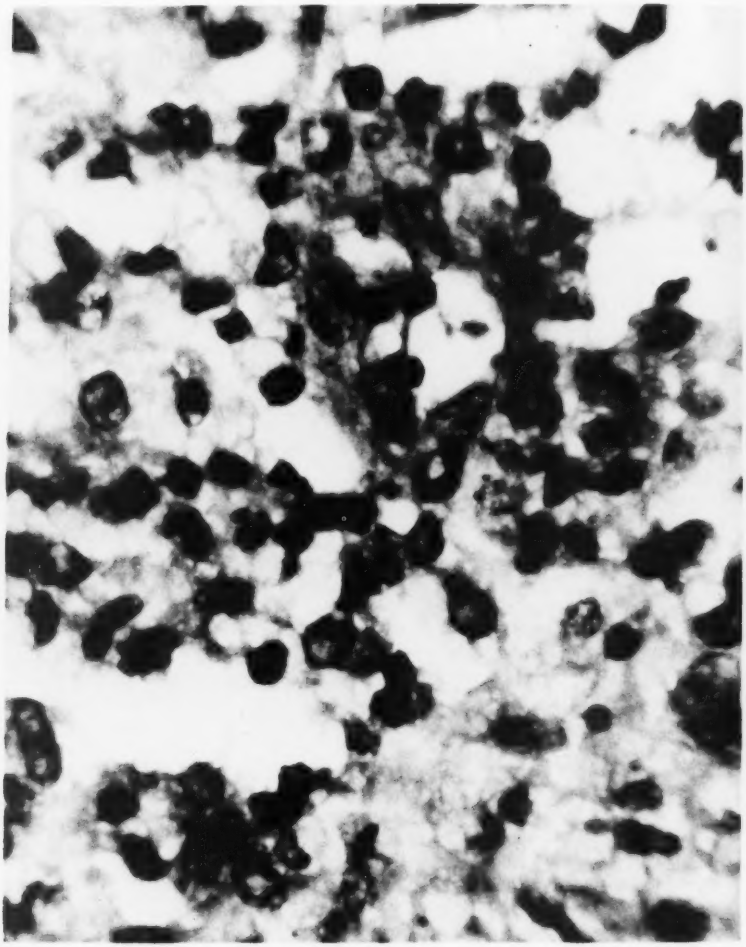

Figure 3. Photomicrograph of abscess wall, showing edema and inflammatory cell infiltrate. (Hematoxylin and eosin stain, × 285)

pus and blood, causing asphyxiation. They also stated that death has occurred at the time of insertion of a mouth gag, apparently as a result of acutely obstructing an already diminished air passage.

As Doctor Bruton has mentioned, retropharyngeal abscess occurs most commonly in infants and young children. Pyogenic bacteria set up an inflammation in the nose or pharynx, or occasionally the ears.

The lymph nodes in the upper posterior wall of the pharynx then become infected and suppurate. These nodes generally disappear in a few years, accounting for the rarity of this entity in older children.

Figure 4. Photomicrograph of abscess wall, trachea, and thyroid gland. The trachea and thyroid were not involved by inflammatory infiltrate. (Hematoxylin and eosin stain, × 7)

The chief complications of retropharyngeal abscess are extension retroesophageally into the mediastinum, rupture into the pharynx with aspiration of pus, erosion into the internal carotid artery with fatal hemorrhage, and laryngeal edema. Clinically there may be dyspnea, dysphagia, cervical adenopathy, fever, sore throat, cough, and convulsions.

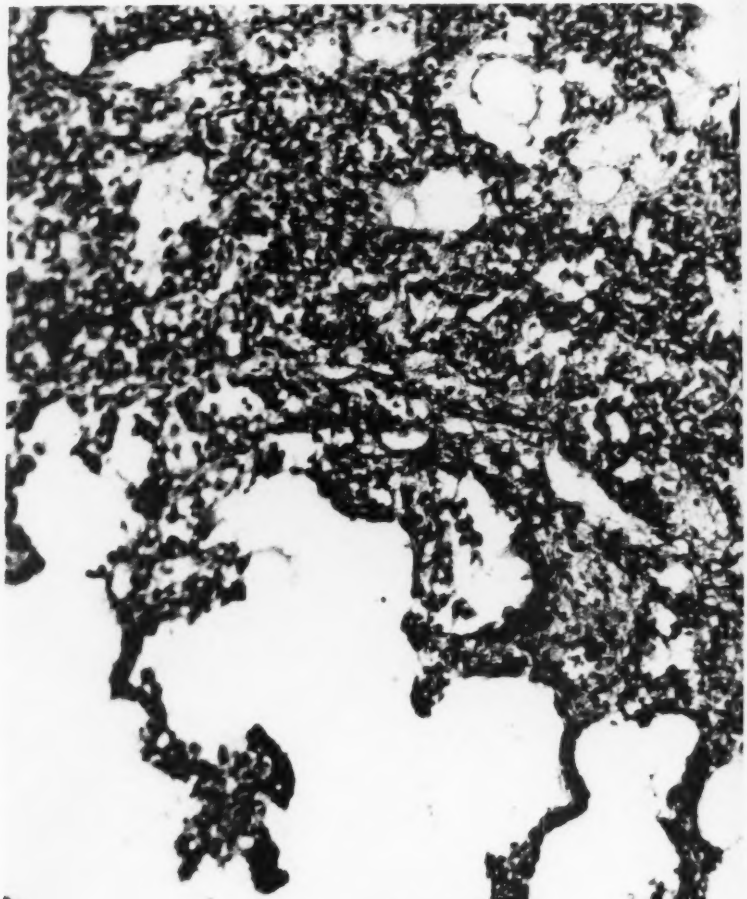

Figure 5. Photomicrograph of lower lobe of the left lung, demonstrating atelectasis and emphysema. (Hematoxylin and eosin stain, × 50)

The diagnosis is made by means of a carefully taken history, direct or indirect (mirror) examination, and by observing forward displacement of the pharynx, trachea, and/or esophagus on a roentgenogram. Prog-

nosis should generally be good. Weille and DeBlois[2] reported 27 cases occurring over a period of 15 years, 13 of which were in children 6 months to 5 years of age. Death occurred in only one case, the result of mediastinitis.

Pathologic diagnoses:

1. Retropharyngeal abscess due to *Staphylococcus aureus*
2. Focal pulmonary atelectasis and emphysema
3. Acute passive congestion of liver and kidneys
4. Meckel's diverticulum

Doctor Delaney: Was the pneumonia that was present in this hospitalized infant in May due to Staphylococcus (Micrococcus)? Were cultures of sputum taken?

Doctor Shuey: There was no entry in the clinical record about sputum cultures having been made.

Doctor Marcarelli: How about the diarrhea for which the infant was admitted in May? Were stool cultures ever made?

Doctor Shuey: The clinical record again does not indicate that this was done. A logical and possible sequence of events could have begun with a staphylococcal enteritis complicated by a staphylococcal pneumonia. These conditions could have been cured by intensive treatment, with a focus of antibiotic-resistant Staphylococci lodging in a cervical vertebra, smoldering for a few weeks, developing into osteomyelitis and breaking through into the retropharyngeal space. Unfortunately we have no proof that this actually took place.

Doctor Rohlfing:* Were sensitivities done in this case?

Doctor Shuey: No results of sensitivity testing of the organism isolated at necropsy are recorded. As the child was given penicillin during the hospitalization, we can assume that this Staphylococcus was not sensitive to the concentration of this antibiotic present in the abscess cavity. That, in itself, does not mean much. However, we know that about 75 per cent of the Staphylococci now being isolated in this hospital are resistant to penicillin in vitro.

Doctor Delaney: This child survived for about 35 minutes after laryngoscopy. With a vasovagal reaction, do you not usually have immediate cessation of the heart beat?

Doctor Shuey: Yes. That mechanism of death seems to be pretty well ruled out in this case.

Doctor Moran:** Was this infant breast-fed? Was there any indication of breast abscess in the mother?

Doctor Shuey: We do not know. If such were the case, it would account for the start of the above postulated sequence of events. The whole

*First Lt. Ronald D. Rohlfing, USAF (MC), Intern, Department of Surgery.
**Capt. James P. Moran, MC, USN, Chief, Obstetrical and Gynecological Service.

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case could then be fitted into an epidemiologic and pathogenetic pattern that has been observed all too frequently since the first recorded observation of the development of penicillin-resistant Staphylococci 9 or 10 years ago.

REFERENCES

1. Jackson, C., and Jackson, C. L. (editors): *Diseases of the Nose, Throat, and Ear.* W. B. Saunders Co., Philadelphia, Pa., 1945. p. 151.
2. Weille, F. L., and DeBlois, E.: Retropharyngeal abscess; problems in treatment. *Arch. Otolaryng.* 39: 344-347, Apr. 1944.

ALCOHOLISM IN AUSTRALIA

"Perhaps the largest and most important group of all alcoholics is made up of normal but rather immature men whose emotional development has been retarded at the "man's man" stage of development. Good army canteen and messmen, good shipmates, good sportings fans, good clubmen, good lodgemen, good mates, good fellows all, they prefer the company and conversation of their own to that of the opposite sex—in their youth without, and later with, the stimulus of alcohol. They behave together much like overgrown schoolboys. They will leave the lounge for the brass rail at licensed establishments, and at mixed parties congregate around the beer keg and the whisky decanter. . . .Coeducation, increased opportunities and increased emphasis on social intercourse between the sexes, civilized mixed drinking at sidewalk cafés, and ritual evening-long dinners with mixed company and good conversation would all help, as would increasing emphasis on the role of the woman in our society—a trend toward matriarchy and the importance placed by the troubador on his lady as important in his life and community affairs."

—WILFRED RICHARDS
in *Medical Journal of Australia*
p. 546, Oct. 12, 1957

AIR FORCE DEDICATES 250-BED HOSPITAL AT CARSWELL AFB, FORT WORTH, TEXAS

A new 250-bed hospital, completed at a cost of $5,200,000, was dedicated in formal ceremonies at Carswell Air Force Base near Fort Worth, Tex., on 24 May. The principal address was given by Major General Dan C. Ogle, Surgeon General of the Air Force, before a crowd of more than 500 military and civilian guests of the air base.

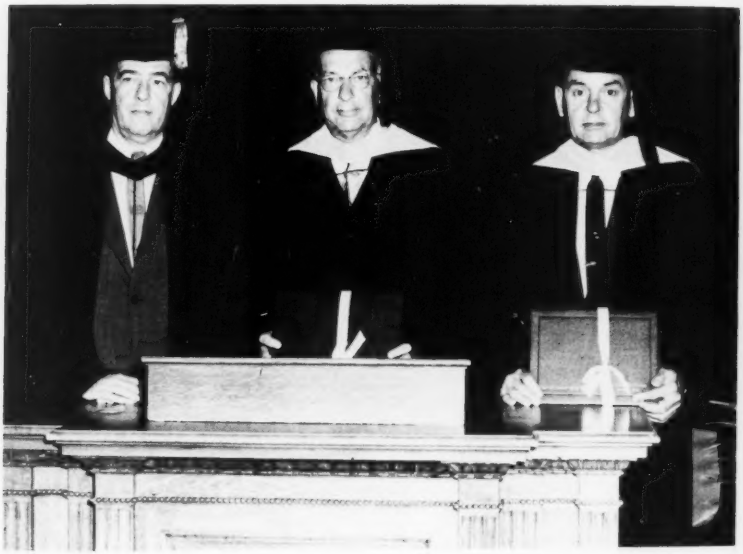
Construction of the new hospital was begun on 12 July 1955 and completed in February of this year. It was informally opened for the admission of patients on 9 March. The building is entirely air conditioned, and is equipped with centralized oxygen and vacuum systems and an audio-visual paging unit. The hospital, which will serve as an orthopedic center as well as treatment facility for the air base, is under the command of Colonel Louis K. Pohl, USAF (MC).

U. S. Air Force Hospital, Carswell Air Force Base, Texas

In addition to General Ogle, the distinguished guests present for the dedication ceremonies included Dr. William E. Flood, President, Tarrant County Medical Society; Dr. Carl A. Gibbe, President, Fort Worth Dental Society; Mr. Clay Berry, President, Fort Worth Chamber of Commerce; Major General John P. McConnell, USAF, Commander, Second Air Force, Barksdale Air Force Base, Louisiana; Brigadier General Nils O. Ohman, USAF, Commander, 19th Air Division, Carswell Air Force Base; and the following senior officers of the Air Force medical service: Brigadier Generals Victor A. Byrnes, James C. Cathroe, Loyd E. Griffis, and M. S. White.

HIGH RANKING MEDICAL OFFICERS GIVEN HONORARY DOCTOR OF SCIENCE DEGREES

Major General Leonard D. Heaton, MC, USA, Commanding General, Walter Reed Army Medical Center, and Rear Admiral Edward C. Kenney, Assistant Chief of the Bureau of Medicine and Surgery for Personnel and Professional Operations, received honorary degrees of Doctor of Science from Denison University, Granville, Ohio, at the 117th annual commencement exercises on 9 June.



Dr. Knapp, General Heaton, and Admiral Kenney

The degrees, conferred by Doctor A. Blair Knapp, President of the University, were accompanied by citations which read in part ". . .to Leonard Dudley Heaton, loyal alumnus, skillful surgeon, eminent hospital administrator, faithful servant of his nation in war and peace . . .and to Edward Christopher Kenney, loyal alumnus, talented physician and medical researcher, eminent hospital administrator, life-long servant of his nation in peace and war. . .the degrees of Doctor of Science."

General Heaton attended Denison from 1919 to 1922 prior to studying medicine at the University of Louisville. Admiral Kenney received a Bachelor of Arts degree in 1926, and subsequently was awarded a Doctor of Medicine degree from the University of Cincinnati.

NEW COMMANDER OF NAVY LABORATORY

Captain John R. Seal, MC, USN, recently assumed command of the U. S. Naval Medical Research Unit No. 3 in Cairo, Egypt. The change of command ceremonies were attended by a group of dignitaries in-

Ambassador Hare, left, and Dr. Seal

cluding United States Ambassador Raymond A. Hare to the United Arab Republic, and Captain Charles S. Moffett, USN, Naval Attache. Captain Seal was formerly head, Communicable Disease Branch, Bureau of Medicine and Surgery.

Correction

The name of Advisory Council on Reserve Affairs to the Surgeon General of the Army was given incorrectly on page 751 of the May Journal in a story concerning the group's first 1958 meeting in Washington.

A. D. A. PRESIDENT MEETS U. S. AND JAPANESE DENTAL OFFICERS DURING TRIP TO FAR EAST

Doctor William R. Alstadt, Little Rock, Ark., President of the American Dental Association, recently visited United States military installations in the Far East after attending the second Asian Dental Congress and the Asian-Pacific Dental Conference in Manila, Philippine Islands.

Left to right, Colonel Burgin, Captain LeClair, Dr. Alstadt, Dr. Sato, Colonel Hosaka, and Colonel Widner

During a six-day tour of Japan on the invitation of the Japan Dental Association, he was guest of honor at a banquet at the U. S. Army Medical Center, Camp Zama, near Yokohama, attended by a group of United States and Japanese dental officers. Among those greeting Dr. Alstadt on this occasion were Colonel Howard N. Burgin, DC, USA, Staff Dental Surgeon, U. S. Army in Japan; Captain Victor A. LeClair, DC, USN, Commanding Officer of the U. S. Naval Dental Clinic, Yokosuka; Colonel Glynn B. Widner, USAF (MC), Chief of Dental Services, Fifth Air Force, Fuchu Air Station; Colonel Tsuneo Hosaka, Chief Dental Officer of the Japan Self-Defense Force; and Doctor Kazuo Sato, President of the Japan Dental Association.

A MESSAGE FROM THE A. M. A.

This month the American Medical Association's Bureau of Health Education has prepared a message on the vital subject of the fitness of American youth.

A great deal of attention is being attracted by claims that American youth is soft, and compared with European youth, weak and in poor condition. The conclusions are controversial, but there hardly can be any controversy about the fact that modern living, with all of its conveniences and labor-saving devices, tends to reduce the amount of physical activity performed by most people, including our young people.

Where American youth formerly walked to school, we now have school buses. Practically every family has a car, and very few persons walk as much as a block to do an errand. Baseball, football, basketball, hockey, and other popular sports provide active participation for a few, while millions sit in the spectator seats eating hot dogs, drinking beer or soft drinks, and exerting themselves only enough to suggest killing the umpire.

The President of the United States has called conferences on the fitness of American youth. The first was held at Annapolis in June of 1956 after being postponed from September 1955 because of the President's heart attack. The second conference was held at West Point in September of 1957. Both conferences were attended by selected citizens serving as individuals but representative of medicine, education, public officialdom, the armed services, amateur and professional athletics, the mass communication agencies, and various other groups.

The Annapolis conference recommended the creation of a Council on Youth Fitness at cabinet level, plus a Citizens' Advisory Committee. The President promptly created a council consisting of the Vice-President as Chairman, the Attorney General, and the Secretaries of Agriculture, Defense, Interior, and Health, Education, and Welfare. The second meeting at West Point was participated in by the Council and the Advisory Committee consisting of 100 citizens appointed as individuals. Included among these were six physicians and two dentists, exclusive of public health personnel. The Vice-President retired as Chairman of the Council and was succeeded by the Secretary of the Interior shortly after the 1957 West Point conference.

The purpose of the Council is to stimulate public interest in the fitness of youth and to encourage and help to co-ordinate efforts toward achieving youth fitness through existing official

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From the Council on National Defense of the American Medical Association. The views and opinions expressed are not necessarily those of the Department of Defense. —*Editor*

and voluntary agencies rather than to create a new operating department for fitness. The Director of the Council is Shane MacCarthy, J. D., educator, author, and lecturer, with a dynamic personality and an abundance of personal fitness.

The place of the medical profession in the fitness program is to advise, at the local and state levels, those agencies which are stepping up their programs in response to the Federal stimulus. The medical profession, with the co-operation of educators, can be extremely valuable in keeping the fitness program on a sound scientific basis and out of the hands of fanatics, cranks, or political and commercial self-seekers.

In the absence of any generally accepted valid tests for fitness, the test for success in improving the fitness of American youth must be the practical one of living. Fitness, therefore, must be more than mere muscular or physical competence. It must extend into the mental, emotional, and social facets of living. It cannot be a fixed quantity, but must be adaptable to the needs of the individual. Not every young man can adapt himself to the flying trapeze, nor every girl to the ballet. For whatever object the individual may have in life, he must, with the aid of the best available professional advice, prepare himself to be fit for that achievement.

Journal Seeks Civilian Associate Editor

The *U. S. Armed Forces Medical Journal* is seeking a civilian physician for appointment as Associate Editor, under the provisions of Department of Defense Directive No. 5154.5, 5 January 1957 (and change 1, 21 May 1957), subject: The Armed Forces Medical Publication Agency. A Civil Service grade of up to GS-15, with a top salary of $13,970 per year, has been established for this position, which officially will be that of Deputy Director of the Agency.

Physicians interested in this new position, which will require full-time residence in Washington, D. C., should make informal application prior to 1 August 1958 to the Director, Armed Forces Medical Publication Agency, 2300 E Street, N. W., Washington 25, D. C. Applicants must be United States citizens, not over 40 years of age, and graduates of approved medical schools in the United States or Canada. Other requirements include a one-year accredited internship, at least two additional years of clinical experience, and a minimum of five years in medical editorial work.

OFFICERS CERTIFIED BY SPECIALTY BOARDS

Supplementary Listing

The Surgeons General of the military medical services have announced that the following regular Medical and Dental Corps officers have been certified by the boards indicated since the listings published in previous issues of the *Journal*.

American Board of Orthopedic Surgery

Dana D. Goldthwaite, Capt., USN
John W. Howard, Comdr., USN

American Board of Urology

Prince D. Beach, Lt. Col., USA

American Board of Obstetrics and Gynecology

Crowell T. Daniel, Jr., Maj., USA

American Board of Internal Medicine

Bruce F. Chandler, Maj., USA
John R. Gibson, Lt. Col., USA

Cardiovascular Diseases

James A. Orbison, Lt. Col., USA

American Board of Ophthalmology

Thomas J. Tredici, Capt., USAF

American Board of Surgery

George S. Banning, Jr., Maj., USAF
Garth B. Dettinger, Maj., USAF
Martin A. Pfotenhauer, Jr., Lt. Col., USA

American Board of Oral Surgery

Kenneth W. Hughes, Lt. Col., USA
Roderick L. Lister, Lt. Col., USA

OFFICIAL DECORATIONS

The following awards were recently announced by the Department of the Air Force.

Legion of Merit

Donald E. Hudson, Col., USAF (DC)
Benjamin A. Strickland, Jr., Col., USAF (MC)

Air Force Commendation Medal

Howard B. Nelson, Col., USAF (MSC)

POSTGRADUATE PROFESSIONAL COURSES OFFERED MEDICAL SERVICE OFFICERS

The following Postgraduate Professional Short Courses for Army Medical Service Officers are announced for the second quarter, Fiscal Year 1959. Personnel interested and eligible to attend should apply through channels to The Surgeon General, Department of the Army, Washington 25, D. C. Attention: MEDCM-CG.

Prosthodontics—Letterman Army Hospital, 6-10 October 1958; Walter Reed Army Medical Center, 8-12 December 1958

The purpose of the course is to provide a comprehensive review of the principles and practice of prosthodontics. It is designed for dental officers who are assigned or may be assigned to a dental prosthetic service. It encompasses an evaluation of the prosthodontic problems in the armed services and the utilization of the Central Dental Laboratory as an adjunct service to clinical practice. Examinations, diagnosis, treatment planning, and prognosis for complete and partial dentures are stressed. Special attention will be given to preparation of the mouth for partial dentures to include occlusal, incisal, and cingulum rest preparations, surveying, and designing. Considerable attention is directed to immediate dentures, impressions, master casts, and jaw relationship records for complete and partial dentures. Selection, arrangement, and articulation of artificial teeth with regard to esthetics and phonetics are presented. Table clinics and patient demonstrations will be included, utilizing color television as a teaching aid.

Anesthesiology for Army Nurse Corps Officers—Brooke Army Medical Center, 6-11 October 1958

This course is designed to provide opportunity for nurse anesthetists to appraise and plan for the practice of anesthesia applicable in Army situations. It will cover but not be limited to: new drugs and technics, probable role of the nurse anesthetist in a catastrophe, standard supplies and equipment, problems confronted by the nurse anesthetist in Class I installations, psychologic aspects of anesthesia in relation to the patient and to the nurse anesthetist, the relationship of the nurse anesthetist to total patient care, in-service education, and professional and personal problems.

Forensic Pathology—Armed Forces Institute of Pathology, 6-11 October 1958

This course is designed to provide training in medicolegal medicine to hospital pathologists and officers in charge of laboratories, both in military and civilian practices. An attempt is made to orient the pathologist and assist him in coping with problems that daily arise where legal factors may be involved. The material is presented by selected specialists in the field of legal medicine. In presenting

the program, lectures, panel discussions, slide reviews, demonstrations, and closed circuit TV are used. Some of the subjects covered are: military law, sudden death, obscure homicide, medicolegal autopsy, gunshot wounds, traumatic death, identification, burned bodies, asphyxial deaths, criminal abortion, cutting and stabbing, serology in legal medicine, death by poison, doctor in court, trace evidence, and trauma in tumors and infections. Most of the material discussed is not found in medical textbooks and periodicals and, therefore, fills a gap in much needed information.

Biostatistics—Walter Reed Army Medical Center, 10-24 October 1958

This course is a portion of the nine-month Military Medicine and Allied Sciences Course which is a sufficiently discrete section to be offered separately. Familiarity with the basic nomenclature and principles of statistics as well as mathematics is a prerequisite. A review textbook reading assignment will be sent to prospective students in advance to help achieve this preparation, and the first two days of the course relates statistical methodology to the design of laboratory experiment and the evaluation of data. This course is intended particularly for those engaged in research, and first priority will be given to students in this category.

Examination for Radioactive Contamination of Food—Walter Reed Army Medical Center, 13 October-5 December 1958

The course has been designed to train military veterinary laboratory officers in methods of detecting and measuring radioactive contamination of foods and in the basic technics for the use of radioactive isotopes as diagnostic and research tools. The scope of the training will cover technics used in assaying different food products for radiocontamination, and to a limited extent, identification of the activity. Sufficient background material will be presented in the fields of mathematics, nuclear physics, tracer chemistry, radiation biology, radiologic safety, and instrumentation to enable the laboratory officer to employ assay technics and to intelligently evaluate specific problems that may arise in the field. Attendance will be limited to laboratory officers who have completed the radiologic health course at the Institute of Nuclear Studies, Oak Ridge, Tenn.

Present Concepts in Internal Medicine—Letterman Army Hospital, 27-31 October 1958

This course will encompass the most recent developments in the field of cardiovascular diseases, general medicine, hematology, gastroenterology, diseases of the chest, and endocrine disorders.

Application of Histochemistry to Pathology—Armed Forces Institute of Pathology, 27-31 October 1958

The course consists of a basic and comprehensive survey of chemical and physical methods that can be used by the pathologist for the study of sections of tissue under the microscope. The subjects

include a review of the theoretical basis of histochemical and classical staining methods, the practical histochemistry of groups of chemical compounds, such as carbohydrates, lipids, pigments, and enzymes, and the histochemistry of particular organs, such as the kidney and pituitary gland. The material will be presented by seminars, laboratory demonstrations, and the study of microscopic slides.

Institute for Army Medical Specialist Corps Officers—Walter Reed Army Medical Center, 2-8 November 1958

This institute is planned as a workshop and will provide an opportunity for AMSC officers to discuss not only trends and developments within their specific professional specialties, but also current Army policies, regulations, and procedures pertaining to the Corps. The scope of the institute will include common sessions directed toward increasing awareness and understanding of the human relations factors in organizational effectiveness, leadership, group participation, and communication.

Nursing in the Medical Management of Mass Casualties—Walter Reed Army Medical Center, 3-8 November 1958

This course is designed to acquaint nurses with the medical aspects and problems of nuclear warfare, to familiarize them with the multidisciplined approach to the situation and to provide them with an opportunity to explore together ways in which the Army Nurse Corps may prepare for the expanded role expected of nursing personnel in national defense. Present concepts and principles of the medical management of mass casualties will be highlighted.

James C. Kimbrough Urological Seminar—Brooke Army Medical Center, 3-5 November 1958

This course will include recent advances in clinical urology and the basic sciences as applied to the field of urology. The presentation of subjects of general urologic interest, based on original work or clinical material, will be encouraged. About 10 per cent of the course time will be allocated to the presentation of interesting or unusual case reports and x-ray films.

Management of Mass Casualties—Brooke Army Medical Center, 1-5 December 1958

The purpose of the course is to indoctrinate officers of the military medical services in current concepts on the management of casualties resulting from the employment of nuclear weapons. The course provides up-to-date information of an unclassified nature on the medical aspects of nuclear warfare, to reduce this information to readily accessible

and useful data in the solution of problems that might arise, and to stimulate thinking on ways of coping with the problem of casualties that might result from nuclear weapons employment.

Institute for Executive Officers— Walter Reed Army Medical Center, 1-6 December 1958

This institute will provide appropriate training for officers currently assigned or expected to be assigned as executive officers in Army hospitals or in the office of Army Command Surgeons. Training will comprise such fields as hospital organization and administration; programming and budgeting; the Hospital Command Management System; preparation of operating plans and in-service education programs; conference technic; review, analysis, and evaluation of operating results; management functions; preparation of hospital directives and reports; integrated data processing; staff and public relationships; discussion of frequently recurring problems; policies regarding care of military dependents; organization and functions of OTSG and Army Surgeon offices.

Pathology of Diseases of Laboratory Animals—Armed Forces Institute of Pathology, 8-12 December 1958

The course is designed to provide training for professional officers who are responsible for the recognition and interpretation of lesions in experimental animals, or have charge of procurement and maintenance of animal colonies. It particularly is intended to help them interpret natural diseases which may negate experimental results or influence the supply of laboratory animals or their suitability for experimental use. It is aimed especially at the needs of various Army, Navy, and Air Force laboratories, most of which have veterinary officers in charge of their colonies. Pathology will be the theme of the course, but this facet will be used as a point of departure for discussion of etiology, diagnosis, and control of the diseases under consideration. Special attention will be paid to those conditions that may affect the suitability of laboratory animals or may alter or interfere with experimental results. The course will be of value only to those individuals qualified to understand disease processes and to absorb information in the field of pathology. Veterinary pathologists should find the course of particular benefit, but pathologists, veterinarians, and others with similar professional backgrounds also will find the course of value.

Pediatric Nursing—Walter Reed Army Medical Center, 8-13 December 1958

This course is designed to provide an opportunity for nurses to appraise and plan for needs of children for comprehensive nursing care applicable in Army situations. Emphasis will be placed on recent developments and trends in clinical aspects of the pediatric care of children suffering from medical and/or surgical disease conditions.

DEATHS

COLE, Olena Margaret, Lieutenant Colonel, AMSC, USA, of Centerville, Calif.; stationed at William Beaumont Army Hospital, El Paso, Tex.; graduated in 1931 from McMurray College, Jacksonville, Ill.; commissioned a second lieutenant in the Army of the United States and ordered to active duty 1 April 1943; commissioned a captain in the United States Army 19 December 1947; died 16 April 1958, age 48, at William Beaumont Army Hospital of a gunshot wound of the head.

HIRSCHMANN, Victor Robert, Colonel, MC, USA, of St. Paul, Minn., stationed at Madigan Army Hospital, Tacoma, Wash.; graduated in 1933 from the Medical College of South Carolina, Charleston, S. C.; commissioned a first lieutenant in the United States Army Reserve 28 August 1934; ordered to active duty 15 November 1934; commissioned a first lieutenant in the United States Army 12 December 1935; died 25 April 1958, age 47, at Madigan Army Hospital of carcinoma of the rectum.

LEVINE, Herbert Jason, Captain, USAF, (MC), of Newton, Mass.; assigned to the 7510th U. S. Air Force Hospital, APO 240, New York, N. Y.; graduated in 1954 from the University of Vermont College of Medicine, Burlington, Vt.; commissioned a first lieutenant in the U. S. Air Force Reserve 18 April 1956; ordered to active duty 3 January 1957; died 24 April 1958, age 29, at Welwyn Garden City, Hertfordshire, England, as the result of an automobile accident.

McCARTHY, Betty Alice, Captain, ANC, USAR, of East Rutherford, N. J.; stationed at the U. S. Army Hospital, Fort Dix, N. J.; graduated in 1930 from St. Mary's Hospital School of Nursing, Passaic, N. J.; commissioned a second lieutenant in the Army of the United States and ordered to active duty 27 April 1942; died 31 January 1958, age 50, at Walter Reed Army Hospital, Washington, D. C., of multiple myeloma.

PREECS, Ethelyn McIntosh, Major, ANC, USA, of Jasonville, Ind.; Walter Reed Army Hospital, Washington, D. C.; graduated in 1937 from the San Antonio State Hospital School of Nursing, San Antonio, Tex.; commissioned a second lieutenant in the United States Army 9 December 1940; died 8 May 1958, age 43, at Walter Reed Army Hospital of postoperative cardiac arrest.

SINCLAIR, Evelyn Hewes, Captain, USAF (NC), of Kennett Square, Pa.; stationed at U. S. Air Force Hospital, Bolling Air Force Base, Washington, D. C.; graduated in 1944 from the Chester County Hospital School of Nursing, West Chester, Pa.; commissioned a second lieutenant in the Army of the United States 8 January 1945; ordered to active duty 15 January 1945; released to inactive duty 12 April 1946; commissioned a first lieutenant in the U. S. Air Force reserve 17 May 1951; ordered to active duty 16 July 1951; died 19 May 1958, age 34, at U. S. Air Force Hospital, Bolling Air Force Base, of cardiovascular collapse.

WILLIAMS, Eloise Beth, Captain, USAF (NC), of Whiting, Iowa; stationed at the U. S. Air Force Hospital, Offutt Air Force Base, Nebr.; graduated in 1934 from the St. Joseph Mercy Hospital School of Nursing, Sioux City, Iowa; commissioned a second lieutenant in the United States Army Reserve 21 June 1943; ordered to active duty 21 June 1943; transferred to the United States Air Force Reserve 1 July 1949; died 1 May 1958, age 46, at the U. S. Air Force Hospital, Offutt Air Force Base, of a cerebral hemorrhage.

WILSON, Gladys Helen, Captain, ANC, USA, of Notasulga, Ala.; stationed at U. S. Army Hospital, Ft. Benning, Ga.; graduated in 1941 from the Columbus City Hospital School of Nursing, Columbus, Ga.; commissioned a second lieutenant in the Army of the United States 12 March 1942; ordered to active duty 20 March 1942; commissioned a first lieutenant in the United States Army 10 March 1948; died 1 May 1958, age 43, at Walter Reed Army Hospital, Washington, D. C., of carcinoma of the breast.

Reviews of Recent Books

CHEMISTRY OF LIPIDES AS RELATED TO ATHEROSCLEROSIS, A Symposium, compiled and edited by *Irvine H. Page*, M. D. 342 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $8.50.

This volume is a publication of a symposium attended by investigators of the metabolism and chemistry of lipids.

Fascinating speculations and interesting problems, but few solutions, are presented. The nature of this volume is best shown by listing phrases which abound in it: "Only a beginning has been made. . . fragmentary knowledge. . .unsolved problem. . .challenging and complex problem. . .has not been explained. . .these data cloud the validity." It is apparent that the reader will harvest a crop of question marks, not answers.

A printed volume intended to be helpful should represent distilled, logically presented observations and interpretations, conclusions and recommendations for definitive action. It is questionable whether a report of any symposium is worth putting into print because presentations and discussions are usually diffuse and speculative; experts prefer to discuss challenging problems, controversial aspects, and theories rather than widely accepted conclusions. This book is no exception. Therefore, although the dust jacket states the volume is intended to appeal particularly to "those in all types of food industry, physicians interested in blood vessel disease and epidemiologists," it is unlikely to be of value to such persons or indeed to any practicing physician, regardless of specialty. In brief, this book is a glorified type of "minutes" of a meeting.

—*ROBERT J. HOAGLAND, Col., MC, USA*

LABORATORY MEDICINE—HEMATOLOGY, by *John B. Miale*, M. D. 735 pages; 192 illustrations and 9 plates, including 5 in color. The C. V. Mosby Company, St. Louis, Mo., 1958. Price $13.75.

This is taken from the Foreword by W. A. D. Anderson: "Laboratory medicine has lagged behind its true role in medical practice in both the general education of physicians and in the evaluation of clinical pathology as a specialty of medical practice." From the Preface by Doctor Miale, the author: "This book on Hematology and two others in preparation on Chemical Pathology and Microbiology emphasize the correlation between laboratory and clinical data. It ranges from basic mechanisms to the interpretation of laboratory data in diagnosis and therapy. Only tests which are ordered with discrimination, performed with skill, and interpreted with understanding yield significant data."

The book is, in general, a rather sophisticated presentation of the physiology and physiopathology of the formation, function, and destruction of blood elements. It is a commendable attempt to provide a coherent and contemporary background for the practice and inter-

pretation of hematologic laboratory procedures. It is up to date as of 1955 with a few references in some sections as late as 1956.

It is not a book without flaws. Overall, it gives the impression of a work of scholarship rather than experience, and the author's attitude seems sometimes to be uncritical. The text has many minute errors of fact which do not seriously detract from its worth. Some of the illustrations are not good. The color plates of the blood and marrow cells are the worst this reviewer has seen. Diagrams such as "Fig. 87, The major factors concerned in erythropoiesis," and "Fig. 99, Catabolic pathways of hemoglobin" are overly generalized and inaccurate. "Fig. 20, Aspiration of sternal marrow" demonstrates the needle lodged in the sternum rising out of a great bushiness of unshaved chest hair in disregard of the most elementary preparation for a surgical procedure. The photomicrographs of the red cells generally lack contrast or definition or both.

These are minor disparagements of a valuable book. It is indeed the best of its kind.—*WILLIAM H. CROSBY, Lt. Col., MC, USA*

FAT CONSUMPTION AND CORONARY DISEASE: THE EVOLUTIONARY ANSWER TO THIS PROBLEM (A basic approach to the prevention and arrest of coronary disease), by *T. L. Cleave*, M. R. C. P. (Lond.). Foreword by *Percy Stocks*, C. M. G., M. A., M. D., F. R. C. P., F. S. S. 40 pages. Philosophical Library, New York, N. Y., 1958. Price $2.50.

This small, 40-page monograph outlines a new concept correlating coronary disease with fat consumption. The author applies the Darwinian theory of evolution and the natural law of adaptation to explain an instinct of appetite. He proposes the principle that an organism may rely on this instinct to tell it what it should eat, but only as long as the instinct is being exercised on natural substances; that is, on substances occurring in the natural environment of the organism.

In civilized countries the instinct of the appetite fails because of the many unnatural substances, especially concentrated fats as butter, chocolate, and shortening, and concentrated sugars. Combinations of these unnatural substances he calls "arbitrary food mixtures," and the consumption of the unnatural fats is offered as the cause of coronary atherosclerosis. The author also gives a brief discussion on the proper selection of diet to prevent this disease.

—*DOSS O. LYNN, Col., MC, USA*

FUNDAMENTALS OF NEUROLOGY, by *Ernest Gardner*, M. D. 3d edition. 388 pages; illustrated. Published by W. B. Saunders Co., Philadelphia, Pa., 1958. Price $4.75.

Textbooks may be provocative or narcotizing. Often a little of both appears. Gardner's *Fundamentals of Neurology* is such a book. This neatly bound, clearly printed, 388-page volume, in its third edition, strives valiantly to introduce the author's conception of neurologic principles to an audience of undergraduates including nurses, physiotherapists, occupational therapists, and medical students.

Physiology and anatomy of the nervous system are discussed, with brief excursions into pathology and clinical neurology. Homely analogies are freely used, on occasion effectively. Most of the photographs are superb and many line drawings help to clarify the text. Each chapter is briefly summarized and followed by short biographies of investigators mentioned therein. Chapter references are few but well chosen and nicely described by the author. A glossary and an adequate index complete the book.

An astonishingly detailed chapter on microscopic anatomy of the nervous system makes the gross anatomy presentations appear glaringly scanty. An excellent chapter on excitation and conduction contrasts sharply with the confusing discussion of the forebrain. Throughout the book such inequalities occur. At times too elementary, the book is often beyond the author's avowed audience. Few nurses could read this book and gain more than a blurred image of the nervous system. For a medical student it is neither an anatomy or physiology text. This reviewer indeed found the good chapters provocative but the vague ones narcotizing. The book is only recommended for serious students having access to well-stocked medical libraries.

—HENRY W. HOGAN, Maj., MC, USA

THE PRACTICE OF INFECTIOUS DISEASE, by *Louis Weinstein*, Ph. D., M. D. 501 pages. Landsberger Medical Books, Inc., New York, N. Y. Distributed by the Blakiston Division of the McGraw-Hill Book Co., New York, N. Y., 1958. Price $8.50.

The preface of this book contains the inference that the practice of infectious disease is being so changed by the use of chemotherapy that past concepts need to be modified. Advertised as a "handbook" for general practitioners, it is a volume of short essays. No attempt at easy reference or special cross-indexing has been made, which has become the generally accepted form of a handbook for busy practitioners. The first three chapters are short essays on the principles of the treatment of infections, which set the tone of the book as the new approach to infectious disease. Other chapters are a discussion of infectious disease by anatomic region and organ systems in the form of short essays, many too brief to be much more than dictionary descriptions, and the final one on fever of obscure origin dwindles off into an outline and ends in platitudes. The book is easy to read but does not fulfill in a convincing form the thesis stated in the preface. It certainly is not a handbook for ready reference for the general practitioner. The book is poorly bound, and contains frequent typographic and occasional editorial errors, *i. e.*, on page 66, line 4: ". . .to kill Anophelene species of mosquitos. . . ."

—WILLIAM D. PRESTON, Col., USAF (MC)

DRUGS OF CHOICE (1958-1959), edited by *Walter Modell*, M. D. 931 pages; illustrated. The C. V. Mosby Company, St. Louis, Mo., 1958. Price $12.75.

This is a remarkably good book. The editor has assembled the written thoughts of 37 clinical specialists concerning drugs used in their

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fields. Each chapter is laconic, readable, and full of information which is personalized and at times superficial.

It will serve a useful purpose in every hospital library where the house staff can review the clinical uses of drugs in a particular field in a matter of minutes. The book contains too little pharmacology to be useful to medical or graduate students.

In my opinion there is only one drawback to the book: There is no true need for it. The scope of this book is, at present, adequately handled by the *New and Nonofficial Drugs*. Personally, I cannot abide books as expensive as this which announce that they will be obsolete within 18 months following the purchase of the book.

—*EDWARD J. HUYCKE, Capt., MC, USA*

HEART DISEASE IN INFANCY AND CHILDHOOD, by *John D. Keith*, M. D.; *Richard D. Rowe*, M. B., F. R. C. P.; and *Peter Vlad*, M. D. 877 pages; illustrated. The Macmillan Company, New York, N. Y., 1958. Price $22.50.

The authors of this comprehensive text on the most recent knowledge of heart disease in children have drawn freely from the world literature and their own 20 years of experience in the cardiac section of the Hospital for Sick Children at Toronto, Ontario. In one five-year period they studied more than two thousand patients with heart disease, the majority having congenital malformations.

The first chapters include an excellent discussion of physical diagnostic methods, and describe the embryology of congenital and acquired cardiac defects. Subsequent chapters cover the incidence, pathology, hemodynamics, electrocardiography, cardiac catheterization, angiography, diagnosis, prognosis, and treatment of congenital cardiac disease.

The last section of the book discusses cardiovascular manifestations and associated findings in acquired heart disease. Included among these are rheumatic fever, collagen disorders, arrhythmias, myocarditis, pulmonary heart disease, glycogen storage disease, and the effects of thyroid, electrolytes, anemia, and renal and neuromuscular disorders on the heart. There also are good discussions of the cardiac disorders in mongolism, Marfan's syndrome, cardiac tumors, gargoylism, and congestive failure.

Three appendices contain a concise discussion of pre- and postoperative care, a table of pertinent drugs and dosages, and a fold-out chart summarizing the clinical features and treatment of congenital heart disease. One significant error was detected; on page 852 the digitalizing dose of digoxin is given as 0.3 mg per lb instead of 0.03 mg per lb.

This book is clearly written, well organized, and adequately illustrated. Each chapter is supplemented by an up-to-date bibliography. It should be of great value to every general practitioner, pediatrician, internist, cardiologist, and medical student.

—*ANDREW M. MARGILETH, Comdr., MC, USN*

BONE DISEASES in Medical Practice, by *I. Snapper*, M. D. 229 pages; with 24-page section of 48 plates. Grune & Stratton, Inc., New York, N. Y., 1957. Price $15.

This authoritative text is divided into 31 chapters. The leading chapter deals with the physiology of bone and the second short one on the radiologic examination. The subsequent chapters, with the exception of the last, deal with the difficult diseases in which bone and other tissues are involved. Each disease is discussed in adequate chronological sequence, including complications. The last chapter deals with differential diagnosis of bone disease. It is a comprehensive and informative 22 pages. The index is followed by 48 pages of radiologic prints produced with very good fidelity. Each chapter is followed by appropriate references. A number of chapters and tables are included. Although the index may be considered as rather brief by some readers, in general, it appears to be adequate.

This should be a valuable reference volume to the internist, pediatrician, endocrinologist and those engaged in isotope technics.

—*FRANCIS W. PRUITT, Col., MC, USA*

PRACTICAL CLINICAL CHEMISTRY, A Guide for Technicians, by *Alma Hiller*, Ph. D. 265 pages. Charles C Thomas, Publisher, Springfield, Ill, 1957. Price $6.50.

This second edition of this monograph presents a glossary of 26 biochemical procedures in clinical diagnostic technics that covers the usual routine requests made by physicians. Three chapters are devoted to general laboratory procedures, standardizations of reference solutions, and photometry. As in the previous edition, each determination is presented initially in detail, and in an outline operational form which may be detached from the book if desired. This is advantageous as these can be removed, placed in transparent covers and used by the technicians performing the procedures. The procedures presented are well established and accepted through common usage, several have been slightly modified in the interest of simplicity and economy of time without sacrifice of accuracy. This book is of value to those working in or directing an average diagnostic laboratory. The extent of revision in this edition, however, would not appear to justify replacing a copy of the first edition.—*WILLIAM H. LEE, Lt. Col., USAF (MSC)*

TUMORS OF THE SOFT SOMATIC TISSUES, A Clinical Treatise, by *George T. Pack*, M. D., LL. D., F. A. C. S., and *Irving M. Ariel*, M. D., F. A. C. S. 820 pages; 652 illustrations. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, N. Y., 1958. Price $30.

This volume is a very comprehensive reference on tumors of the soft tissues. It is written by Doctors Pack and Ariel and a group of 15 collaborators, and is based upon 25 years of work and a total of 717 cases of these tumors. Technically the book is well prepared and bound. The type is legible and errors are at a minimum.

The soft somatic tissues are defined by the authors as consisting of connective tissue, fat, muscle, fascia, synovia, blood and lymph

vessels, and nerve tissue. Each chapter is profusely illustrated with drawings, charts, and photographs. There is a bibliography at the end of each chapter and an index at the end of the book.

This book is well written and well organized, as it must be to cover such a wide field. The recommended therapies are based primarily on the personal experience of the authors, and alternative methods are described and appraised. As a matter of interest, the over-all 5-year survival reported is 39 per cent.

Altogether this is an excellent reference that fills a definite gap in the libraries of those interested in the treatment of tumors.

—PHILIP D. CRONEMILLER, Capt., MC, USN

THERAPEUTIC HEAT, Volume II of Physical Medicine Library, edited by *Sidney Licht*, M. D. 466 pages; illustrated. Elizabeth Licht, Publisher, New Haven, Conn., 1958. Price $12.

Therapeutic Heat is the second volume of the series to be known as the Physical Medicine Library and is a welcome addition to the growing list of textbooks on physical medicine and rehabilitation.

This volume offers a comprehensive coverage of the subject of heat, presenting all aspects of its clinical uses. The great variety of therapeutic apparatus and methods for applying heat are described. The physiologic effects, technics of application, dosage, and indications and contraindications for the modalities in clinical use are clearly presented and should be of value to clinicians in general. In addition, there are individual chapters devoted to the use of heat in rheumatic diseases, orthopedics, peripheral vascular diseases, neuromuscular disorders, pelvic diseases, and diseases of the eye.

For the physiatrist and student of physical medicine who needs to extend his knowledge of the technical and biophysical principles involved, it is gratifying to find several chapters devoted to detailed presentations of the physics of heat, the biophysics of diathermy, microwave, ultrasound, and thermometry.

The book is a valuable contribution and should be of real value to all physicians practicing and teaching in the field of physical medicine and rehabilitation. *—ANIELLO F. MASTELLONE, Lt. Col., MC, USA*

THE TREATMENT OF FRACTURES, by *Lorenz Böhler*, M. D. Volume III. Translated from the Thirteenth German Edition by *Alfred Wallner*, M. D., and *Otto Russe*, M. D. 2307 pages; 1699 illustrations. Grune and Stratton, Inc., New York, N. Y., 1958. Price $21.

This elaborate textbook is the fifth edition in English and comprises 802 pages with 1,699 illustrations. Interestingly, all three volumes total 2,307 pages, which is over twelve times the size of the first edition on fractures published in 1929. The author has related 30 years' experience in the management of these injuries, and the work was of particular interest to the reviewer inasmuch as he enjoyed a postgraduate course at Professor Böhler's *Unfallkrankenhaus* in 1955.

Impressions gained at that time of characteristic German efficiency, adherence to established procedure, and infinite attention to detail are reflected in this textbook.

There is an impressive statistical analysis of all the injuries from punch-card records of 90,000 inpatients and 656,000 outpatients, as well as roentgenograms of 256,000 cases. Typical case histories are used to emphasize teaching points, and Doctor Böhler's wide experiences in the treatment of war wounds (two world wars) are recorded. Essentially he emphasizes the same principles of treatment as in his first edition. These include accurate anatomic reduction, use of the nonpadded plaster, sound wound surgery, avoidance of open reduction, and use of the extremity during immobilization. Repetition and irrelevant verbosity detract from the volume, and insufficient attention has been given to modern methods of open reduction and internal fixation, modern treatment of shock, use of antibiotics, and newer anesthesia modalities for an authoritative textbook. Each section has a list of "Questions We Should Ask Ourselves to Avoid Failures With This Method." This is often obvious and tedious, but always interesting. The appendix deals with education, organization, and economic significance of traumatic surgery. The reader is given a splendid review of the accident problems in Austria and many of the thoughts can be applied to our own efforts in this country.

The material should be of interest to all clinical surgeons, especially those engaged in military orthopedic and traumatic surgery. The volume ranks with Watson-Jones as a classic in fracture treatment.

—EARL W. BRANNON, Col., USAF (MC)

ULCERATIVE COLITIS, by *Harry E. Bacon,* B. S., M. D., Sc. D., LL. D., F. A. C. S., F. A. P. S. Foreword by *Alton Ochsner,* B. A., M. D., Sc. D., F. A. C. S., F. A. P. S. (Hon.), with contributions by *Paul T. Carroll,* B. S., M. D. Chapter on Anesthesia by *Leroy W. Krumperman,* M. D. 395 pages; 184 illustrations. J. B. Lippincott Co., Philadelphia, Pa., 1958. Price $15.

This book is based on the author's experience with 440 cases of ulcerative colitis over a 17-year period, and incorporates a substantial review of the literature. Every facet of the problem is thoroughly discussed. As one would expect, Dr. Bacon devotes considerable space to discussion of surgical treatment, including ileostomy, but it is noteworthy that one of the longest and best chapters is concerned with nonsurgical therapy.

The study of this perplexing disease must necessarily reveal diversities of opinion and experience. Dr. Bacon records these faithfully and without bias. The only apparent prejudicial note concerns those psychiatrists who incarcerate the patient for long periods of psychotherapy, "frequently ignoring toxic symptoms and irreversible changes."

The text material is written in an informal, easily readable style. Rhetorical questions are used effectively to introduce controversial

topics. Particularly commendable are the photographs and listings of ileostomy appliances and accessories, including prices and addresses of manufacturers or distributors.

There is little room for criticism of this book. There are a few minor errors in the text material, certainly not excessive for a first edition. There is some duplication which might be eliminated. Some points of discussion are repeated, perhaps for emphasis, in subsequent chapters. A few photographs are reproduced twice. The placement of references at the end of each chapter results in multiple listing of reference material.

These imperfections are actually quite minor and do not detract from the excellence of this work. It is highly recommended for anyone who is interested in the ulcerative colitis patient in any stage of his disease. —*JAMES H. STEWART, Comdr., MC, USN*

THE YEAR BOOK OF UROLOGY (1957-1958 Year Book Series), edited by *William Wallace Scott*, M. D., Ph. D. 359 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $7.50.

This issue of the "Year Book of Urology" has again maintained its excellent presentations of the world literature on this subject. The selected articles are worth reading, and very little extraneous material is included in the synopses from the original work. The chapter headings are the same as in previous years, making it convenient to review entire subjects. The author's notes at the end of some of the abstracts are very interesting and at times express an opinion entirely different than what was stated in the original paper.

It is gratifying to see more of the foreign literature included in this year's volume. The subject and author indexes at the end of the book are well arranged for easy and rapid reference identification.

This book is a must for the busy urologist who does not have the time to go through all of the allied medical journals on urology, where many excellent articles on both clinical and research subjects are presented. It is a must for the resident who is preparing for his specialty boards, as the material presented is up to date, while most standard texts are several years out of date when first published.

—*NICHOLAS MALLIS, Maj., MC, USA*

FUNDAMENTALS OF ELECTROCARDIOGRAPHY AND VECTORCARDIOGRAPHY, by *Laurence E. Lamb*, M. D. 142 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1957. Price $9.50.

This monograph contains the basic fundamentals of electrocardiography and vectorcardiography. The opening chapter deals with fundamental vector concepts for which simple mathematical explanations are given. The next five chapters lay the solid groundwork for the chapters on the normal electrocardiogram and fundamentals of vectorcardiography. The use of the triaxial reference system and the more utilitarian hexaxial reference system is clarified. This is further inte-

grated into a spatial reference system by addition of the transverse reference system. Clarification of the mean spatial QRS-T angle is afforded together with a simple functional "road-mileage" type of spatial QRS-T angle chart, which can be mathematically proved and has been of inestimable value to me in studying borderline tracings. The clinical section is devoted to the problems of cardiac enlargement, conduction defects, pericarditis, myocardial infarction and arteriosclerotic heart disease, drugs and metabolism, arrhythmias and their interpretation. The book is laced with spatial vectorcardiograph models and related tracings, which do not require a magnifying glass to read.

A perusal of this text serves to answer the query as to what vectorcardiography offers that is superior to routine electrocardiography. In problem and borderline electrocardiograms, the use of the vectorcardiogram and force analysis of the standard tracings often give a clue as to the presence or absence of pathology. For those who profess an interest in cardiology and who feel a need to comprehend the flux of literature on vectorcardiography, this book is commended. It is recommended that this book also be used as a reference for hospital libraries. —*ARCHIE A. HOFFMAN, Col., USAF (MC)*

WORLD-ATLAS OF EPIDEMIC DISEASES, Part III, Second of four issues, edited by Professor Dr. med. *Ernst Rodenwaldt* and Professor Dr. med. *Helmut J. Jusatz*, Heidelberg, under the sponsorship of the Heidelberger Akademie der Wissenschaften. 32 pages; 10 maps. Valk-Verlag, Hamburg 1, Germany, 1958. Price DM 63, plus extra charge for postage and packing.

This latest issue of the World Atlas, like the previous ones, presents a text in English and German concerning the clinical, statistical, and geographic descriptions of specified diseases, and the maps which comprise the atlas. As in previous issues, the pages are not serially numbered but fit into the loose-leaf binder provided for Part III. Cholera (1931-1955) and Bacterial Dysentery in Asia (1900-1954) are the contributions to regional epidemiology. For Diphtheria (1934-1953), Poliomyelitis (1947-1955), Smallpox (1949-1955), Filariasis in Man (1955), and Trachoma (1930-1955), the scope is world-wide. The plague pandemic of the twentieth century is documented and illustrated. Another significant addition to the climatological factors affecting populations, their health and welfare, is included; the global regions of thermic sultriness relate as usual to altitude.

The authors provide extensive bibliographies for each subject, attesting to the thoroughness with which they have searched for reliable information. At the same time, they are careful to define the limitations of their data. The work continues to be concise, lucid, and precise.

The reader agrees wholeheartedly with those who have reviewed previous issues of this great work that it will make a most complete and authoritative reference. It is essential to the activities of public

health physicians, military medical officers, medical faculties and students, as they contemplate the past and present in terms of disease and human ecology and as they make their plans for further studies and control efforts.—*SIDNEY A. BRITTEN, Capt., MC, USN*

HIGH ARTERIAL PRESSURE, by *F. H. Smirk,* M. D., F. R. C. P., F. R. A. C. P. 764 pages; illustrated. Charles C Thomas, Springfield, Ill., 1958. Price $15.

High Arterial Pressure is among the most comprehensive and thorough volumes ever written on the subject. The author is well qualified to present the material covered in this book and he does so in an extremely readable style.

The physiology, pathogenesis, pharmacology, and treatment of high arterial pressure are given complete coverage, with treatment being given prime consideration. The author describes his approach to the evaluation of the patient, his disease, and treatment. He brings out the latest developments concerning all phases of the disease and presents his own views as well as those from the literature. The bibliography at the conclusion of each section is extremely complete and invaluable by itself.

This book is a very important contribution to the medical literature and is strongly recommended for all medical libraries. It will prove invaluable to the general internist and is a requisite for the cardiologist. Surgeons and general practitioners will find it useful for reference in the treatment of the hypertensive patient.

WILLIAM R. SCHILLHAMMER, Maj., MC, USA

AUTONOMIC IMBALANCE AND THE HYPOTHALAMUS. Implications for Physiology, Medicine, Psychology, and Neuropsychiatry, by *Ernst Gellhorn,* M. D., Ph. D. 300 pages; illustrated. University of Minnesota Press, Minneapolis, Minn., 1957. Price $8.50.

In this monograph Dr. Gellhorn describes in some detail many of his experiments in which he has studied the sensitivity of the hypothalamus and the interaction between the hypothalamus and the autonomic nervous system and the central nervous system. They are presented in detail for workers who are research oriented. He discusses his concept of "tuning" of the sympathetic and parasympathetic branches of the autonomic nervous system and the way in which this state of being sympathetically "tuned" or parasympathetically "tuned" alters the response to subsequent stimuli. Dr. Gellhorn's ideas about the clinical implication of his work are exciting, and bring us close to the hope of correlation between brain function and mental processes. He suggests that altered mental functions are related to definite alterations in neurophysiologic functions and that it may soon be possible to measure these alterations. There are many diagrams and an extensive bibliography of 330 references. This monograph will be of interest to all investigators in the field of psychophysiology. Clinicians will be interested in the summary and suggested clinical applications. —*ROBERT L. WILLIAMS, Lt. Col., USAF (MC)*

THE MEDICAL MANAGEMENT OF CANCER, by *Henry D. Diamond,* M. D., F. A. C. P. 179 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1958. Price $6.75.

The author, a member of the staff of the Sloan-Kettering Institute for Cancer Research and the Memorial Hospital, has drawn from the experience in cancer diagnosis and therapy possible at such an institution, together with a careful review of the literature.

For purposes of presentation the author has divided cancer into types for which the presently accepted mode of therapy is medical or nonsurgical, and those types treated by surgical means. The first category contains the malignant lymphomas, the leukemias and plasma cell myeloma.

Despite the fact that at present there is no really satisfactory treatment for any cancer (except perhaps those of the skin, which can be treated by large doses of x-radiation), there are certain presently accepted forms of therapy which are beneficial, at least, in the sense of amelioration of many of the symptoms. This relatively small work admirably covers the highlights and many of the details of the symptoms, signs, diagnostic tests, and therapy of the malignancies discussed. The role of the internist in the management of cancers, in which primary treatment is surgical, is also well covered. A short but adequate appendix on intracavitary application of radioisotopes is included.

This book is recommended to physicians who are concerned with the care of patients with cancer. The cancer specialist might find certain subjects for which he would desire a more extensive discussion of certain areas of controversy which are covered only in part. However, the large amount of material included in such a brief work makes this a readable and valuable book.

—*NATHANIEL I. BERLIN, Lt. Comdr., MC, USNR-R*

CURRENT THERAPY 1958, edited by *Howard F. Conn,* M. D. 827 pages. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $12.

There are 297 contributors to this book, most of whom are well-known authorities in their particular field. The consulting editors are outstanding teachers and clinicians.

The purpose of this volume is to give the practicing physician authoritative and current information on medical therapy. This has been achieved in an excellent, clear, and concise manner. Most of the newer drugs and technics of therapy are dealt with in a practical and helpful way—outlining both principles and details of therapy. It is reassuring to see that the recommendations for such drugs as the steroids and tranquilizers are kept in proper perspective by most contributors.

The contents include 16 sections which deal with infectious diseases, diseases of the various organ systems, of metabolism, allergic

diseases, obstetric and gynecologic conditions, and diseases due to physical and chemical agents.

In a book of this type there may be disagreement among physicians as to selection of drugs or dosages used for various conditions. In one instance, for example, digitalis leaf may be used in cardiac arrhythmias in the dosage of 0.2 to 0.3 grams (3 to 4½ grains) daily. No mention is made of possible digitalis toxicity if this dosage is continued. If the book is used as a guide only, these discrepancies are of a very minor nature.

As an excellent and quick reference to up-to-date treatment, this volume can be highly recommended for the practicing physician.

—DOSS O. LYNN, Col., MC, USA

THE CHEMICAL DYNAMICS OF BONE MINERAL, by *William F. Neuman* and *Margaret W. Neuman.* 209 pages; illustrated. The University of Chicago Press, Chicago, Ill., 1958. Price $5.

The book is a personalized record of the authors' attempt to discern relationships among a diversity of data pertaining to the physiology of bone. Those who have an interest in this area of research, may find here some point of reference for their own pursuit of a synthesis of the available knowledge concerning the nature and physiologic interactions of bone mineral. Approximately one fifth of the book is devoted to a review of the physical chemistry of solutions, with emphasis on the behavior of calcium and phosphate under physiologic conditions. The functional significance of ion *activity,* as contrasted with *concentration,* is amply explained and documented. Other of the topics which receive major attention are the interaction between calcium and protein, the state of calcium and phosphate in serum, and the effect of citrate upon the solubility of bone mineral.

The authors hypothesize that parathyroid hormone and vitamin D act synergistically to mobilize bone calcium, by way of an increased accumulation of citrate. This scheme postulates that parathyroid hormone inhibits or destroys coenzyme II, thereby preventing the oxidation of the glycolytic products of carbohydrate metabolism, and that vitamin D enhances the conversion of pyruvate and lactate to citrate. *—THADDEUS J. DOMANSKI, Col., USAF (MSC)*

PRACTICAL PEDIATRICS, by *R. Cannon Eley,* M. D., and *Benjamin Kramer,* M. D. 307 pages. Landsberger Medical Books, Inc. Distributed by The Blakiston Division of McGraw-Hill Book Co., New York, N. Y., 1958. Price $7.

This volume is one of a series of handbooks for the general practitioner. The purpose of this text is to describe accepted pediatric practices and procedures which may be readily acquired and used by the general practitioner. This aim is well carried out. The material presented is accurate and is enjoyable reading. Many practical problems are introduced and discussed in a not too didactic manner so that they are readily understood by the general practitioner without

formal training in pediatrics. There are very few illustrations, graphs, or tables. The chapter on fluid and electrolyte balance could have been presented more simply and a few of the chapters were quite brief; however, these were in the minority. The chapters on "The Newborn," "Premature Infant," and "Infant Feeding" were exceptionally informative.

In summary, the material discussed in this text is accurate, succinct, and forms a practical review of the everyday problems met by the physician who cares for pediatric patients.

—*ERNST R. MOELLER, Capt., MC, USN*

THE YEAR BOOK OF THE EAR, NOSE AND THROAT AND MAXILLOFACIAL SURGERY (1957-1958 Year Book Series). The Ear, Nose and Throat, edited by *John R. Lindsay*, M. D. Maxillofacial Surgery, edited by *Dean M. Lierle*, M. D. and *William C. Huffman*, M. D. 383 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $7.50.

This is the first year that the abstracts of the literature on ear, nose, and throat have been separated from the *Year Book of Eye, Ear, Nose, and Throat* and made into a separate volume. Included with these abstracts is a section on maxillofacial surgery. This gives the book greater value to the specialist in otorhinolaryngology. The ophthalmologist and the otolaryngologist have been gradually separating in their interests. At the same time there has been increasing interest for the ear, nose and throat specialist in maxillofacial surgery because of the increasing frequency of deformities of the face following vehicular accidents. This year book is of great value to the otorhinolaryngologist in that it gives a concise, well-edited summary of the literature that has been presented in this field during the year. The editors present their material in the outstanding manner that has given the year books their well-deserved reputation. The volume is recommended for the otolaryngologist, and any others interested in this field. —*HENRY S. MURPHEY, Col., MC, USA*

THE EARLY DIAGNOSIS OF THE ACUTE ABDOMEN, by Sir *Zachary Cope*, B. A., M. D., M. S. Lond., F. R. C. S. Eng. 11th edition. 188 pages; illustrated. Oxford University Press, New York, N. Y., 1957. Price $4.50.

This small volume first appeared 37 years ago and is now in its eleventh edition. It has 36 figures and 8 reproductions of x-rays, and is well made, easily handled, and readable. The material is presented under 21 different headings with a certain amount of repetition occuring. The author has few references and no bibliography. Some new sections have been added and relate to vascular disease and to gastrointestinal obstruction in the newborn.

Parts of this book are valuable because of the correlation between anatomy and the location of the inflammatory lesion, and between physiology and the location of the obstructing lesion. A refreshing emphasis, regrettably uncommon to this degree in American literature, is put on observations the physician makes with his own eyes, ears,

and hands. The importance of the history and physical and the small but indicative clues to be gotten from these two diagnostic tools are stressed to the partial exclusion of the possibly too popular American prop to diagnosis, the laboratory. Repeatedly the reader is urged to strive for earlier diagnosis of acute abdominal conditions. Statistics are cited to show that in acute abdominal disease the recovery rate is proportional to the earliness of the diagnosis of the condition. Some material presented is archaic or rare enough to not warrant the time spent on it. Examples are the concern with typhoid fever and tuberculosis of the bowel; conversely though, mesenteric lymphadenitis isn't mentioned as a cause of the acute abdomen.

—DON S. WENGER, Col., USAF (MC)

A MANUAL OF ELECTROTHERAPY, by *Arthur L. Watkins*, M. D. 259 pages; 167 illustrations and 1 plate in color. Lea & Febiger, Philadelphia, Pa., 1958. Price $5.

The title of this manual may be misleading because the term "electrotherapy," as used here, is meant to include all physical agents employed in physical therapy that have electricity as their basic form of energy. This book revises the excellent *Manual of Physical Therapy* by the late Doctor Richard Kovacs, and contains an added chapter on the physics and use of ultra sound. Information on the physics of radiant energy that is indispensable for the basic understanding of the use of physical agents in therapy also is presented, and the section on electrodiagnosis should clarify a poorly understood part of physical medicine.

This manual is an excellent reference text for all physicians, especially those not in the specialty of physical medicine.

—GEORGE E. VAUPEL, Capt., MC, USN

PROCEEDINGS OF THE SIXTH INTERNATIONAL CONGRESS OF THE INTERNATIONAL SOCIETY OF HEMATOLOGY, Boston, August 27-September 1, 1956. Prepared and edited by the Publications Committee of the Sixth International Congress of Hematology. 930 pages; illustrated. Grune and Stratton, Inc., New York, N. Y., 1958. Price $25.

The Sixth International Congress of the International Society of Hematology was the largest held to date. The clinical material, therapeutic approaches to hemopoietic problems, immunohematology, and irradiation illness, to mention a few subjects that were discussed, represented the thinking and efforts of specialists in the field of blood diseases in some twenty-eight countries. The "meat" of these contributions is contained in this publication. Considering the scope and extent of the papers presented, the Publications Committee had a difficult task in determining what should be published in full.

This volume contains a wealth of hematologic data that probably will not be published in any other form. It is recommended as a source book of information, both clinical and investigative, for hematologists and those closely related to this field and for internists and surgeons presented with clinical hematologic problems.

—JOSEPH H. AKEROYD, Lt. Col., MSC, USA

New Books Received

Books received by the *U. S. Armed Forces Medical Journal* are acknowledged in this department. Those of greatest interest will be selected for review in a later issue.

MODERN TRENDS IN GASTRO-ENTEROLOGY (Second Series), edited by *F. Avery Jones*, M. D., F. R. C. P. Foreword by *C. F. W. Illingworth*. 439 pages; illustrated. Paul B. Hoeber, Inc., Medical Book Department of Harper and Brothers, New York, N. Y., 1958. Price $16.

REHABILITATION OF THE CARDIOVASCULAR PATIENT, by *Paul Dudley White*, M. D.; *Howard A. Rusk*, M. D.; *Philip R. Lee*, M. D.; and *Bryan Williams*, M. D. 176 pages; illustrated. McGraw-Hill Book Co., Inc., The Blakiston Division, New York, N. Y., 1958. Price $7.

PROGRESS IN ARTHRITIS, edited by *John H. Talbott*, M. D., and *L. Maxwell Lockie*, M. D. 456 pages; illustrated. Grune and Stratton, Inc., New York, N. Y., 1958. Price $12.50.

CARDIOVASCULAR DISEASES, by *David Scherf*, M. D., F. A. C. P., and *Linn J. Boyd*, M. D., F. A. C. P. 3d edition (revised and enlarged). 829 pages; illustrated. Grune and Stratton, Inc., New York, N. Y., 1958. Price $17.75.

OBJECTIVE APPROACHES TO TREATMENT IN PSYCHIATRY, by *Leo Alexander*, M. D. American Lecture Series, Publication Number 327, A Monograph in American Lectures in Objective Psychiatry, edited by *W. Horsley Gantt*, M. D. 139 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $4.50.

THE YEAR BOOK OF ORTHOPEDICS AND TRAUMATIC SURGERY (1957-1958 Year Book Series), edited by *Edward L. Compere*, M. D., F. A. C. S., F. I. C. S. With a Section on Plastic Surgery, edited by *Neal Owens*, M. D., F. A. C. S., F. I. C. S. 463 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $7.50.

THE YEAR BOOK OF DERMATOLOGY AND SYPHILOLOGY, (1957-1958 Year Book Series) edited by *Rudolf L. Baer*, M. D., and *Victor H. Witten*, M. D. 492 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $8.

LIFE INSURANCE AND MEDICINE, The Prognosis and Underwriting of Disease, edited by *Harry E. Ungerleider*, M. D., F. A. C. P., and *Richard S. Gubner*, M. D., F. A. C. P. 994 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $16.50.

THE INTROVERT, A Psychiatric Study of Social Adjustment for Student and Layman, by *Ainslie Meares*, MBBS. B, AGR. SC. DPM. 147 pages. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $4.50.

THE PRESERVATION OF YOUTH; Essays on Health, by *Moses Ben Maimon (Maimonides)*. Translated from the original Arabic (Fi Tadbir As-Sihha) and with an introduction by *Hirsch L. Gordon*, M. D., Ph. D., D. H. L. 92 pages. Philosophical Library, Inc., New York, N. Y., 1958. Price $2.75.

ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, Vol. 68, Art. 3, Pages 657-1266, April 24, 1958. Editor in Chief, *Otto V. St. Whitelock;* Managing Editor, *Franklin N. Furness;* Associate Editors, *Margaret P. Cameron* and *Francis S. Stahl.* "Comparative Clinical and Biological Effects of Alkylating Agents," Consulting Editor *David A. Karnofsky.* 609 pages; illustrated. The New York Academy of Sciences, New York, N. Y., 1958.

ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, Vol. 72, Art. 5, pages 215-226, April 15, 1958. Editor in chief, *Otto V. St. Whitelock;* managing editor, *Franklin N. Furness.* "Half Life of Rhenium and Ages of Minerals," by *Stanley N. Naldrett.* 11 pages; illustrated. New York Academy of Sciences, New York, N. Y., 1958.

ABORTION IN THE UNITED STATES, A Conference Sponsored by the Planned Parenthood Federation of America, Inc., at Arden House and The New York Academy of Medicine. Edited by *Mary Steichen Calderone,* M. D., M. S. P. H. Introduction by *M. F. Ashley Montagu.* 224 pages; illustrated. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, N. Y., 1958. Price $5.50.

DISCOVERING OURSELVES, A View of the Human Mind and How It Works, by *Edward A. Strecker,* M. D., Sc. D., and *Kenneth E. Appel,* M. D., Sc. D., in collaboration with *John W. Appel,* M. D. 3d edition. 303 pages; illustrated. The Macmillan Company, New York, N. Y., 1958. Price $4.75.

BIOLOGY OF NEUROGLIA, compiled and edited by *William F. Windle,* Ph. D., Sc. D. 340 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $8.50.

PSYCHOENDOCRINOLOGY, edited by *Max Reiss,* M. D., D. Sc. 208 pages; illustrated. Grune and Stratton, Inc., New York, N. Y., 1958. Price $7.

THE CLINICAL PHYSIOLOGY OF PHYSICAL FITNESS AND REHABILITATION, by *Ernst Jokl,* M. D. 194 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $8.50.

SCIENCE AND PSYCHOANALYSIS, Volume I, Integrative Studies, edited by *Jules H. Masserman,* M. D. 201 pages; illustrated. Grune and Stratton, Inc., New York, N. Y., 1958. Price $5.75.

TEACH THEM TO LIVE, A Study of Education in English Prisons, by *Frances Banks,* M. A. (Psych.), with a foreword by The Rt. Hon. Lord *Birkett.* 287 pages. International Universities Press, Inc., New York, N. Y., 1958. Price $5.

TUBERCULOSIS IN ANIMALS AND MAN, A Study in Comparative Pathology, by *John Francis,* D. Sc., M. R. C. V. S. 357 pages; illustrated. The Williams and Wilkins Company, Baltimore, Md., 1958. Price $18.

THE PSYCHIATRIC HOSPITAL AS A SMALL SOCIETY, by *William Caudill.* 406 pages; illustrated. Published for The Commonwealth Fund by Harvard University Press, Cambridge, Mass., 1958. Price $6.50.

THE WILL TO LIVE, by *Arnold A. Hutschnecker,* M. D. New revised edition. 292 pages. Prentice-Hall, Inc., Englewood Cliffs, N. J., 1958. Price $4.95.

PRACTICAL CARDIOLOGY, by *Albert Salisbury Hyman,* M. D., F. A. C. P., F. A. C. C. 307 pages. Landsberger Medical Books, Inc. Distributed solely by The Blakiston Division of the McGraw-Hill Book Co., Inc., New York, N. Y., 1958. Price $7.

DIAGNOSTIC MEDICAL PARASITOLOGY, by *Edward K. Markell,* Ph. D., M. D., and *Marietta Voge,* M. A., Ph. D. 276 pages; 115 figures, including 5 in color. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $7.

PROCEEDINGS OF THE SIXTH INTERNATIONAL CONGRESS OF THE INTERNATIONAL SOCIETY OF HEMATOLOGY, Boston, Mass., August 27-September 1, 1956. Prepared and edited by the *Publications Committee of the Sixth International Congress of Hematology.* 930 pages; illustrated. Grune & Stratton, Inc., New York, N. Y., 1958. Price $25.

NEW FRONTIERS IN CHILD GUIDANCE, J. H. W. van Ophuijsen Memorial Volume. Edited by *Aaron H. Esman,* M. D. 218 pages. International Universities Press, Inc., New York, N. Y., 1958. Price $4.

THE FIRST BOOK OF WORLD WAR I, by *Louis L. Snyder.* 96 pages; illustrated with maps by *Leonard Derwinski.* Franklin Watts Inc., New York, N. Y., 1958. Price $1.50.

AUSCULTATION OF THE HEART, by *Abe Ravin,* M. D. 166 pages; illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1958. Price $6.

DISEASES OF THE THYROID AND PARATHYROID GLANDS, by *Bernard J. Ficarra,* A. B., Sc. B., M. D., D. S. Foreword by *Arnold S. Jackson,* M. D. 295 pages; illustrated. Intercontinental Medical Book Corporation, Publishers and Booksellers, New York, N. Y., 1958. Price $8.50.

THE ADMINISTRATION OF HEALTH EDUCATION AND PHYSICAL EDUCATION, by *Jesse Feiring Williams,* M. D., Sc. D.; *Clifford Lee Brownell,* Ph. D.; and *Elmon Louis Vernier,* Ed. D. 5th edition. 387 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $4.75.

MICROBIOLOGY AND EPIDEMIOLOGY, by *LaVerne Ruth Thompson,* R. N., M. A., M. S. in Public Health. 4th edition. 581 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $6.

ANOMALIES OF INFANTS AND CHILDREN, by *D. McCullagh Mayer,* D. D. S., M. D., F. A. C. S., F. I. C. S., and *Wilson A. Swanker,* M. D., F. A. C. S., F. I. C. S. 454 pages; illustrated. McGraw-Hill Book Company, Inc., The Blakiston Division, New York, N. Y., 1958. Price $12.

MANUAL FOR THE APHASIA PATIENT, by *Mary Coates Longerich,* Ph. D. Foreword by *J. M. Nielsen,* M. D. 277 pages; illustrated. The Macmillan Company, New York, N. Y., 1958. Price $4.75.

A TEXTBOOK OF ORAL PATHOLOGY, by *William G. Shafer,* B. S., D. D. S., M. S.; *Maynard K. Hine,* D. D. S., M. S.; and *Barnet M. Levy,* A. B., D. D. S., M. S. 714 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $15.

CLINICAL PERIODONTOLOGY, The Periodontium in Health and Disease (Recognition, diagnosis and treatment of periodontal disease in the practice of general dentistry), by *Irving Glickman,* B. S., D. M. D., F. A. C. D. 2d edition. 978 pages; illustrated with 720 figures. W. B. Saunders Co., Philadelphia, Pa., 1958. Price $17.50.

ANOMALIES OF INTESTINAL ROTATION AND FIXATION (Including Mesentericoparietal Hernias), by *Roberto L. Estrada,* B. Sc., M. D., C. M., D. Surg. (McGill), F. R. C. S. (C), F. A. C. S. 161 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $6.50.

NERVES EXPLAINED, A Straightforward Guide to Nervous Illnesses, by *Richard Asher,* M. D., F. R. C. P. 157 pages. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $2.75.

MEMOIRS OF A GOLDEN AGE, by *Maurice Davidson.* 142 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $3.75.

THE CLINICAL APPLICATION OF PROJECTIVE DRAWINGS, by *Emanuel F. Hammer,* Ph. D. 663 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $13.50.

THE MEASUREMENT AND APPRAISAL OF ADULT INTELLIGENCE, by *David Wechsler.* 4th edition. 297 pages; illustrated. The Williams and Wilkins Company, Baltimore, Md., 1958. Price $5.

ELECTROCARDIOGRAPHIC ANALYSIS, Volume 1—"Biophysical Principles of Electrocardiography," by *Robert H. Bayley,* M. D. 237 pages; illustrated. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, N. Y., 1958. Price $8.

PRINCIPLES OF RESEARCH IN BIOLOGY AND MEDICINE, by *Dwight J. Ingle,* B. S., M. S., Ph. D. 123 pages. J. B. Lippincott Company, Philadelphia, Pa., 1958. Price $4.75.

CARE OF THE PREMATURE INFANT, by *Evelyn C. Lundeen,* R. N., and *Ralph H. Kunstadter,* M. D., F. A. C. P., F. A. A. P. 367 pages; 87 illustrations. J. B. Lippincott Company, Philadelphia, Pa., 1958. Price $8.

HISTORY OF DENTAL LABORATORIES AND THEIR CONTRIBUTIONS TO DENTISTRY, by *Robert J. Rothstein.* 277 pages; illustrated. J. B. Lippincott Company, Philadelphia, Pa., 1958. Price $7.50.

SKIN GRAFTING, by *James Barrett Brown,* M. D., and *Frank McDowell,* M. D. 3d edition. 411 pages; 328 figures and 6 color plates. J. B. Lippincott Company, Philadelphia, Pa., 1958. Price $15.

THE MANAGEMENT OF CHILDHOOD ASTHMA, by *Frederic Speer,* M. D. 116 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $4.75.

PHYSICS FOR THE ANAESTHETIST, Including a Section on Explosions, by Sir *Robert Macintosh,* D. M., F. R. C. S. E., F. F. A. R. C. S., M. D. (hon. causa) Buenos Aires and Aix-Marseilles; *William W. Mushin,* M. A., M. B., B. S., F. F. A. R. C. S.; and *H. G. Epstein,* M. A., Ph. D., F. F. A. R. C. S. Illustrated by Miss *M. McLarty* and Miss *M. Beck.* 2d edition. 443 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $15.50.

SOLDIER IN WHITE, by *John M. Gibson.* 277 pages. Duke University Press, Durham, N. C., 1958. Price $6.75.

PEDIATRIC INDEX, A Guide to Symptomatological Diagnosis and Current Management, by *Edwin F. Patton,* M. D. 639 pages. The C. V. Mosby Co., St. Louis, Mo., 1958. Price $13.50.

MEDICAL SOCIOLOGY, Theory, Scope and Method, by *Norman G. Hawkins,* B. Ed., Ph. D. 290 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $6.75.

SIGNS, SYMPTOMS AND TREATMENT OF CERTAIN ACUTE INTOXICATIONS, by *William B. Deichmann,* Ph. D., and *Horace W. Gerarde,* M. D., Ph. D. 2d edition. 154 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $3.75.

FUNDAMENTALS IN CARDIOLOGY, by *John B. Wild,* M. D. 83 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1958. Price $4.50.

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